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مكتبة ادهم

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Urinary Tract Diseases

Urinary tract diseases

Symptomatology of urinary tract diseases

{1} Pain :

[A] Renal pain :

Character : Dull aching pain.
Site : In the renal angle (angle between the sacrospinalis and the last rib) referred anteriorly to the anterior renal point (at upper outer quadrant of the abdomen).
Due to : Pelvic distension or stretch of renal capsule.

[B] Ureteric colic :

Character : Colicky pain.
Site : Usually extends from the loin to the groin.
Due to : Obstruction by a stone. As the stone moves downwards, the pain is felt in the groin and it may radiate to the upper thigh and to the penis in males or the vulva in females (along ilio-inguinal nerve). When the stone is impacted at lower end of the ureter, there is severe pain referred to tip of the penis.

[C] Vesical pain :

Character : Varies from slight suprapubic discomfort to severe agonizing pain.
Site : In the suprapubic region. It is referred to the tip of penis with severe desire to micturate with the expulsion of only a few drops of blood stained urine (strangury).
Due to : Inflammation, stone, or bladder carcinoma.

[D] Prostatic and seminal vesical pain :

Site : The pain is deeply seated in the pelvis & is usually referred to the perineum, but may be referred to the suprapubic region or to one iliac fossa.
Due to : Inflammation.

[E] Urethral pain :

Character : This is burning pain during micturition.
Due to : Inflammation or stone.

{2} Hematuria :

Definition : Passage of blood in urine.

- **It varies from** frank hematuria to smoky urine with microscopical hematuria.
- **This should be differentiated from red coloured urine** which may occur secondary to hemoglobinuria, the intake of beetroots or some drugs as furadantin or rifampicin.

Hematuria can be classified into :

[1] Total hematuria :
Means the passage of blood all over the stream of urine.
This usually indicates that it comes from the kidney or from massive vesical bleeding.

- Hematuria from the kidney is usually associated with cylindrical clots.
- Hematuria from the bladder is associated with discoid clots.

[2] Terminal hematuria :
Means the passage of blood at the end of micturition.
This usually indicates that the pathology is in the bladder or posterior urethra.

- Sometimes , senile prostatic enlargement (prostatic piles) is the cause.

[3] Initial hematuria :
Means the passage of blood at the beginning of the act of micturition.
This usually indicates a urethral origin.

Differential diagnosis is by three glasses test

Causes of hematuria :

General causes :

1. Blood diseases e.g. thrombocytopenic purpura or haemophilia.
2. Anticoagulant therapy e.g. heparin.
3. Hypertension.

Items	Trauma	Inflammation	Stone	Tumor	Others
Renal Causes	Accidental , Post-operative & After instrumental procedures	Acute glomerulonephritis, Acute pyelonephritis & TB ulcer	Stone	Neoplasms of kidney	**Congenital** e.g. polycystic kidney. **Vascular** e.g. infarction of the kidney.
Ureteric Causes	After instrumental procedures	Ureteritis	Stone	Neoplasms of ureter	
Bladder Causes	Trauma	Bilharzial cystitis , Non-specific cystitis & Tuberculous cystitis	Stone	Neoplasms of bladder	
Prostatic Causes		Tuberculosis or Bilharziasis		Prostatic cancer	Senile prostatic enlargement
Urethral Causes	Rupture urethra	Urethritis	Stone	Papilloma & Carcinoma	

{3} Pyuria :

Means the presence of pus in urine. It may be microscopic or gross.

{4} Chyluria :

The urine looks milky due to the presence of chyle. The colour clears on addition of ether. Addition of Sudan III gives an orange colouration.

{5} Phosphaturia :

The urine looks turbid due to the presence of phosphate crystals. Adding few drops of acetic acid clears the turbidity.

{6} Necroturia :

Pieces of tissue appear in urine. They represent necrotic tissue of bladder carcinoma.

{7} Pneumaturia :

The term means the presence of air in urine. It may be due to a colovesical fistula, infection with gas forming organisms.

{8} Frequency of micturition : This is due to :

1. Irritation of bladder wall e.g. stone or inflammation.
2. Reduction in bladder capacity e.g. by tumours or fibrosis.
3. Prostatic enlargement.
4. Increased urine production by the kidney (polyuria).
5. Stone in the intramural ureter.

• **In benign prostatic hyperplasia the frequency is mainly nocturnal, with bladder stones it is mainly diurnal, while in cystitis it is present day and night.**

{9} Difficulty in micturition :

- This is due to some obstruction in the urinary pathway.
- In benign prostatic hyperplasia, the patient has to relax for few seconds at the beginning of micturition (hesitancy), while patients with a urethral stricture have to strain to pass urine.
- In these patients the urinary stream is weak.

{10} Urinary incontinence :

Definition : Uncontrolled escape of urine.

Types :

1. **True incontinence** is urine loss without warning secondary to defective urethral sphincters.
2. **Stress incontinence** is urine loss in association with physical strain.
3. **Urge incontinence** is involuntary loss of urine with strong desire of micturition.
4. **Overflow (false) incontinence** is loss of urine due to chronic urinary retention or 2ry to a flaccid bladder. The intravesical pressure finally equals the urethral resistance ; urine then constantly dripples.
5. **Enuresis** is bed wetting at night (physiological, functional or organic).
6. **Fistula** abnormal communication between urinary & gynecological organs.

{11} Retention of urine:

Definition : Inability to pass urine in spite of a full bladder.

Causes :

[A] Mechanical causes:

1. Urethra : Rupture , phimosis , urethritis , stricture and stone.
2. Prostate : Prostatitis , abscess , senile enlargement and cancer.
3. Bladder : Bladder-neck obstruction , stone and cancer.
4. Outside the bladder : Pregnancy , fecal impaction and pelvic tumours.

[B] Neurogenic causes may be :

1. Spasmodic : as in post-operative (reflex) and hysterical (functional) retention.
2. Atonic : as in old age , tabes dorsalis , poisoning (e.g. belladonna and arsenic) , fevers (e.g. typhoid) and neurogenic bladder.

Senile prostatic enlargement & postoperative retention are the commonest causes in males

According to its presentation , retention of urine may be :

1. Acute retention : No urine can be passed at all. The patient has a strong desire to pass urine , he has severe pain and the bladder is tender and full.

2. Chronic retention : The patient passes some amount of urine , but residual urine remains in the bladder after each act , gradually filling the bladder.

3. Chronic retention with overflow : With prolonged obstruction, the bladder is continuously full with a large amount of residual urine and the patient passes urine only when the intravesical pressure exceeds the intraurethral pressure.
The actual complaint of the patient is continuous dribbling of urine. The condition is painless and the bladder is not tender but it is dangerous as it leads to back pressure changes on both kidneys.

Differential diagnosis :

From anuria : In retention , the urinary bladder is full and forms a median pyriform swelling in the suprapubic area which is dull on percussion while in anuria the bladder is empty.

Treatment :

[A] Acute retention :

1. Conservative treatment is tried at first : An analgesic is administered. The patient is advised to get out of bed and a warm bath is advised. Except in patients with organic obstruction , an injection of a parasympathomimetic drug is prescribed.

2. Catheterization is attempted if conservative treatment fails. A soft catheter is used **(Nelaton or Foley).**

3. If urethral catheterization fails , **a percutaneous suprapubic catheter** can be inserted under local anaesthesia **(suprapubic cystocath).**

[B] Chronic retention :

- If there are no back pressure effects on the kidneys and biochemical investigations are normal , there is no need for catheterization and the cause of retention should be treated.
- Otherwise , a self retaining Foley catheter should be inserted. **With gradual evacuation of the bladder.**
- Then the cause of chronic retention is investigated and treated.

Investigations for urinary tract diseases

[I] Laboratory investigations :

Urine examination , blood urea & serum creatinine.

[II] Radiological investigations :

[1] Plain X-ray of the urinary tract :

This is sometimes referred to KUB , an abbreviation for kidney , ureters, and bladder.

The following findings are looked for :

1. The soft tissue shadow if the kidney is enlarged with renal swellings.
2. The psoas shadow is obliterated by a perinephric abscess.
3. Bone contour may reveal spina bifida , osteoarthrosis of the lumbar spine.
4. Radio-opaque shadows e.g. stones.
5. Calcification of the urinary tract.

[2] Intravenous pyelography IVP (urography IVU) :

- The idea is to give iodine containing contrast material **(Urographin)** which is concentrated by the kidneys and excreted in urine.
- The patient is asked to micturate and a post-voiding film is taken to demonstrate the amount of any residual urine.

Advantages :

1. Can diagnose most of the congenital, traumatic, inflammatory & neoplastic lesions of the urinary tract.
2. Give a very good idea about the kidney functions.

- **Precautions should be taken against allergic complications.**

Contraindications :

1. Urea above 100mg.
2. Acute infection.

[3] Infusion urography :

In patients with raised blood urea (100-200 mg/100 ml), intravenous urography will not show up. In this situation a contrast material as **urographin** (2 ml/Kg) is added to an equal volume of saline and then infused slowly over a period of 10 minutes. This may produce a useful urogram.

[4] Ultrasound examination :

1. It confirms the site and size of the kidneys.
2. It can diagnose dilatation of the pelvi-calyceal system and the thickness of renal cortex.
3. Calculi are easily visualized.

4. Differentiate between a cystic or a solid lesion of the kidney.
5. Can estimate the amount of post voiding residual urine.

Transrectal ultrasound is now very efficient in visualizing prostatic lesions.

[5] CT scan & MRI

[6] Ascending (retrograde) pyelography :

This is mainly indicated when the data obtained from I.V urography are not conclusive. A cystoscope is introduced and fine ureteric catheters are passed along the ureters to the renal pelvis and the dye is injected directly into the catheter.

Advantages : The test provides good delineation of the pelvi-calyceal system but no information about the renal function.

[7] Cystography :

During the performance of an IVU, the bladder will be visualized in late films **(Descending cystography).**

For better visualization, a catheter is passed and the dye is injected directly in the bladder **(Ascending cystography).**

[8] Urethrography.

[9] Renal arteriography :

Nowadays , it is mainly indicated for the diagnosis of renal artery stenosis and arteriovenous malformations.

[10] Radioisotope scanning.

[11] Endoscopy :

Recent advances in fibreoptic endoscopes have made it possible to visualize every part of the urinary tract with great accuracy. It is now possible to perform urethroscopy , cystoscopy , ureteroscopy and nephroscopy. Any suspicious lesion is biopsied.

[12] Urodynamic studies :

Very useful in patients with obstructive uropathy.

Congenital anomalies

Congenital anomalies of the kidneys

Classification :

<table>
<tr><th>Anomalies of number</th><th>Anomalies of shape, form and fusion</th></tr>
<tr><td>1. Agenesis : Bilateral or unilateral.
2. Supernumerary kidney.</td><td>1. Horse shoe kidney
2. Crossed ectopia : Discoid, sigmoid, L-shaped.</td></tr>
<tr><th>Anomalies of volume and structure</th><th>Anomalies of renal vasculature</th></tr>
<tr><td rowspan="2">1. Multicystic kidney.
2. Polycystic kidney.
3. Simple renal (Solitary) cyst.
4. Hypoplastic kidney.</td><td>1. Aberrant, accessory or multiple vessels.
2. Renal artery aneurysm and A-V fistula (rare).</td></tr>
<tr><th>Anomalies of the renal pelvis</th></tr>
<tr><td>Bifid pelvis (double pelvis) - Pelviureteric junction obst.</td></tr>
<tr><th>Anomalies of ascent</th><th>Anomalies of rotation</th></tr>
<tr><td>Ectopic kidney (pelvic or iliac).</td><td>Incomplete - Excessive - Reverse.</td></tr>
</table>

Anomalies of volume and Structure

Multicystic kidney

- This is usually unilateral, nonhereditary, and is characterized by an irregular lobulated mass of cysts.The ureter usually atretic or absent.
- No function is detectable on urography.
- Nephrectomy is the treatment of choice as neoplastic changes have been noted in it.

Polycystic kidney

Etiology : Failure of fusion between :

1. The secreting units (nephrons) : which develop from the metanephros. &

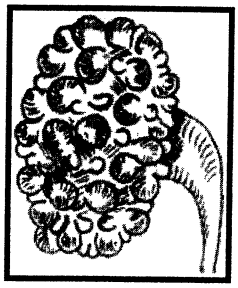
2. The collecting tubules **:** which develop from the mesonephros.

Leading to multiple retention cysts which compress the surrounding renal tissue.

> This may be of the adult or infantile type.

Autosomal dominant polycystic kidney disease (Adult type)

- This is the most common form.
- 50% of cases have associated cysts in the liver, lung, pancreas or other organs.
- 30 to 40% of patients have intracranial aneurysms.



Grossly :

- The kidneys are enlarged.
- Their surfaces are studded with various sized cysts.
- On section, the cysts are scattered throughout the parenchyma.
- The fluid in the cysts is usually clear but may be hemorrhagic or infected.
- The cysts are not communicating with each other **(D.D. hydronephrosis).**

Clinical picture :

- **Typically for the first 10 years :** The kidneys are normal in function and anatomy.
- **From age 10 to 30 years :** US may show the presence of cysts, although patient is asymptomatic.
- **After this, symptoms start in the form of :** Microscopic or gross hematuria, Hypertension, Urinary tract infections, Flank pain, Palpable kidneys or Chronic renal insufficiency.
- **From age 40 to 50 years :** Elevation of creatinine begins.
- **After the age of 50 :** Dialysis or Transplant typically becomes necessary

Investigations : (As general)

1. IVP : The calyces are stretched over the cysts. Giving the **spider leg appearance.**

2. Isotope studies : will reveal multiple **"cold avascular spots"** in large renal shadows.

3. Ultrasonography : Superior to both the above in the diagnosis of polycystic kidneys.

Treatment : Conservative and supportive except for complications :

1. The patient should drink large quantities of **water,** and have **a low protein diet.**

2. Hypertension : should be controlled.

3. Pyelonephritis : must be treated to prevent acceleration of renal damage.

4. Infection of cysts : requires surgical drainage.

5. Hemodialysis or renal transplantation : may be indicated.

6. There is no evidence that excision or decompression of cysts **(Rovsing's operation)** is of any benefit. However, if a large cyst is compressing upper part of ureter or pelvis, it should be aspirated or resected.

Autosomal recessive polycystic kidney disease (Infantile type)

May present by :

1. Enlarged kidneys impeding vaginal delivery.
2. Renal failure in infants.
3. Pulmonary hypoplasia : Newborns with the severe form of the disease die of respiratory disorders within a few days of birth.

Simple renal (solitary) cyst

Etiology :

It is not clear whether this lesion is congenital or acquired. Its origin may be similar to that of polycystic kidneys, or it may be related to tubular obstruction.

Clinical picture :

1. The condition is commonly asymptomatic.
2. Pain.
3. Hemorrhage into a cyst or infection will lead to acute symptoms.
4. Renal mass if the cyst is huge.

D.D. : Neoplastic renal masses.

It is usually possible to differentiate between them by ultrasonography & CT.

- Angiography is an invasive technique that is now rarely necessary.

Management :

[A] If the diagnosis is established :

Simple renal cysts should be left alone, unless infected or compressing the upper ureter.

[B] If diagnosis is not established :

Percutaneous needle aspiration of the cyst is done.

1. If clear fluid is aspirated : It is examined cytologically, and the cyst is then drained and cyst fluid replaced with a sclerosant such as 95% alcohol.

2. If the aspirate contains blood or has a high fat content or with positive cytology :

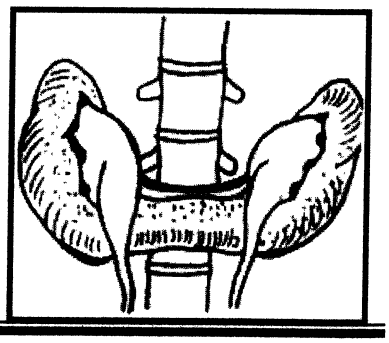
> Surgical exploration should be considered as there is a high risk that the growth is cancerous.

- **Surgical procedure :**
  1. The extra-renal portion of the cyst is excised (Kirwin's method).
  2. Partial nephrectomy.
  3. Total nephrectomy : if there are indications of malignancy.

Horseshoe kidney

Characters :

1. It occurs early in embryologic life when the kidneys lie low in the pelvis.
2. Ascent of horseshoe kidney is arrested by isthmus being blocked against the inferior mesenteric artery.
3. Normal rotation cannot occur and each renal pelvis lies on the anterior surface of its kidney.
4. The lower poles lie nearer to mid line.
5. Ureters are kinked by the isthmus.
6. The blood supply is usually abnormal.
7. The adrenal glands are in their normal positions.



Clinical picture :

- **1/3 of patients :** remain asymptomatic.
- **The rest develop :** calculi, hydronephrosis, infection, and hematuria.
- **A fixed mass** may be palpable below the umbilicus.

Investigation :

1. IVP : The most characteristic finding is presence of lower pole calyces that point medially.
2. Ultrasonography & C.T scan.

Treatment :

1. Asymptomatic cases : no treatment.
2. Pressure effects on ureters : division of the isthmus.
3. Disease of one ½ of kidney : heminephrectomy.

Anomalies of Renal Vasculature

An accessory artery passing to the lower pole of the kidney can cause obstruction or hydronephrosis. More frequently, however, aberrant or accessory vessels accentuate hydronephrosis present due to another reason.

Ectopic kidney:

Due to failure of the kidney to ascend. It presents by a mass in right iliac fossa. In **IVU**, ureter is straight and short.

Mobile kidney

Normally both kidneys move with respiration for 1 inch

Etiology:

Rapid loss of weight.

Clinical picture:

- ✓ May be asymptomatic
- ✓ **Deitel's crisis:** acute loin pain, relieved suddenly with passage of large amount of urine.

IVU: abnormal position with kinked ureter.

Treatment:

- ✓ Conservative (fattening diet)
- ✓ Rarely, surgical nephropexy.

Anomalies of the Renal Pelvis

I- Bifid pelvis

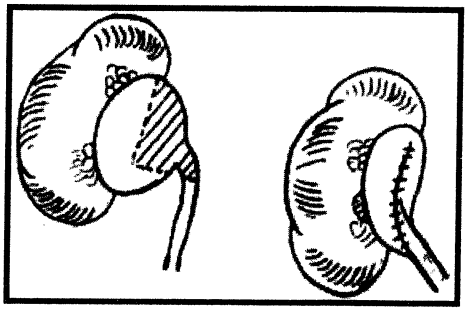
This is the most common anomaly of the upper urinary tract, and is found in about 4% of individuals. It is more common on the left side. A double pelvis is more liable to become the seat of infection, calculus formation or hydronephrosis than a normal pelvis, however, this condition is usually harmless, and is only accidental finding on urography.

II- Pelvi-ureteric junction obstruction

This is a common cause of hydronephrosis in children and adolescents.
Bilateral obstruction occurs in 15% of cases

Treatment : The most popular operation is a pyeloplasty **(Anderson-Hynes operation) :**

- This entails excising the proximal segment of the ureter as well as the redundant pelvis.
- A longitudinal cut is then made in the upper ureter which is anastomosed to lower part of the renal pelvis.



Congenital anomalies of the ureter

Classification :

1. Anomalies of number : Agenesis-Duplication.
2. Anomalies of structure : Atresia and hypoplasia - Megaureter - Ureteral stenosis - Ureteral valves - Ureteral diverticulae.
3. Anomalies of position : Retrocaval ureter.
4. Anomalies of termination : Vesicoureteric reflux- Ectopic ureter - Ureterocele.

Anomalies of Number

Ureteric Duplication (duplex anomaly) :

In incomplete duplication the ureters usually join in the lower third of their course. Complete duplication is less frequent, with both ureters opening separately into the bladder. In this case the ureter draining the upper part opens into the bladder below the ureter draining the lower part of the kidney.

Regarding ectopic ureter:

- Is usually the upper member of a double ureter.
- May open into the posterior urethra or seminal vesicle in the male.
- May open into the urethra or vagina in the female.
- May cause urinary incontinence in females (treated by re-implantation).
- May require transplantation into the bladder.

Anomalies of the bladder

1. Exstrophy (Ectopia vesicae).
2. Persistent urachus : it leads to a urinary fistula at the umbilicus.
3. Contracture of the bladder neck.

Exstrophy (ectopia vesica)

Incidence : 1 in 50.000 live births. It is much more common in males.

Clinical picture :

1. Absent anterior wall of the bladder.
2. Absent anterior abdominal wall from the umbilicus downward.
3. An umbilical hernia is always present and may be associated with indirect inguinal hernias as well.
4. In the male there is associated epispadias with a shortened widened penis.

Complications :

1. Recurrent urinary tract infections.
2. Development of bladder cancer due to metaplastic changes.
3. Excoriation of the skin.
4. Sterility

Treatment :

[A] Urinary diversion (uretero-segmoidostomy) with excision of the bladder, followed by repair of the epispadiac penis.
Disadvantages : The long term problems associated with diversion.
[B] Functional closure : by complete reconstruction is now the first line of therapy.
1. The primary objective in functional closure is to convert the exstrophy to a complete epispadias with incontinence in first 48 hours, anterior or posterior pelvic osteotomy may be required in delayed cases.
2. Penile and urethral reconstruction is then attempted at 2 to 3 years of age.
3. Correction of incontinence and reflux is delayed until bladder capacity has increased.

Contracture of the bladder neck (Marion's disease)

Treatment : consists of bladder neck division or transurethral resection which may cause incontinence in females or later development of retrograde ejaculation in boys.

Anomalies of the urethra

1. Duplication of the urethra.
2. Urethral stricture.
3. Posterior urethral valves.
4. Anterior urethral valves.
5. Urethrorectal and vesicorectal fistulas.
6. Hypospadias.
7. Chordee without hypospadias.
8. Epispadias.

Hypospadias

Definition :

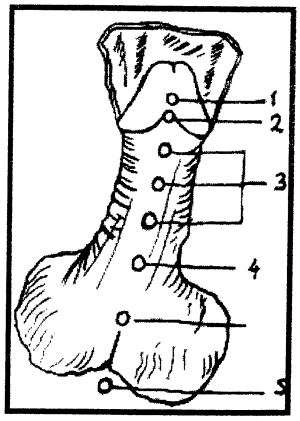
The urethral meatus opens on the under surface of the penis proximal to the tip of the glans penis.
The missing part of the urethra is replaced by a fibrous band (chordae).
The more proximal the meatus, the more apparent will be the ventral chordee (curvature).

Incidence : 1:300 male children.

Classification :

1. Glandular or glanular.
2. Coronal.
3. Penile (anterior, mid, posterior).
4. Penoscrotal.
5. Scrotal.
6. Perineal.

- **Hypospadias is :** Anterior in 65% of cases.
  Midpenile in 15% of cases.
  Penoscrotal or perineal in 20% of cases.



Clinical picture :

1. Abnormal hooded prepuce.
2. Increased incidence of undescended testis and inguinal hernia.
3. The meatus may be stenotic.
4. In cases of posterior hypospadias, exclude the possibility of Intersex.

Treatment :

- **They should not be circumcised** because the preputial skin may be Useful for future reconstruction.
- **For psychologic reasons** hypospadias should be repaired before school age.
- **More recently,** pediatric surgeons have begun repair at 6 to 18 months.
  1. The first step : Straightening the penis by removal of chordee if present.
  2. Second stage : Urethroplasty.

  > One stage repair combining the two steps in one operation is done now.

Epispadias

Incidence of complete epispadias : 1 in 120,000 males and 1 in 450,000 females.

Classification in males :

Glanular, penile, penopubic and complete type.

- **Glandular epispadias** is not associated with incontinence.
- **Penile and penopubic epispadias** are usually associated with incontinence.

Injuries to the urinary tract

Renal injuries

- About 10% of all injuries seen in the casualty department involve the urinary system.
- Renal injuries are the most common of them.

Incidence : The relative rarity of serious renal injury is due to :

1. The kidney is protected by the ribs and by the muscles of the back.
2. The kidneys are mobile.
3. The fibrous capsule.

Etiology :

1. Blunt injuries : The commonest (80%) caused by :

(a) Direct trauma to the abdomen (common) , flank or back.

(b) Indirect or counter coup injury (less common) e.g. due to falling from a height.

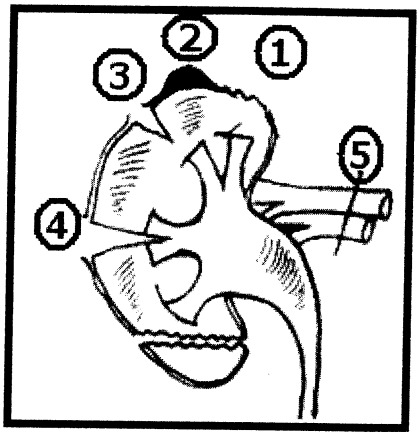
2. Penetrating injuries :

Associated visceral injuries are present in 80% of renal penetrating wounds.

3. Mild trauma may rupture an abnormal kidney, e.g. hydronephrosis or Wilm's tumor.

4. Iatrogenic injuries occur during surgical operations.

Pathology :



[A] Early pathology :

1. Bruising and ecchymosis (most common lesion).

2. Hematoma : Subcapsular hematoma or perirenal hematoma.

3. Incomplete tears of the renal parenchyma :

> If it involves the pelvi-calyceal system , hematuria will develop.

> If it involves renal capsule , a large perirenal hematoma will develop.

4. Complete tear : It extends through the capsule and pelvi-calyceal system with extravasation of blood and urine. Hematuria is marked.

5. Vascular injuries.

6. Associated injuries.

[B] Complications : The following sequelae may occur :

1. Hydronephrosis. Fibrosis of the perirenal tissues leads to ureteral obstruction.

2. Renal Atrophy or fibrosis.

3. Perirenal cyst or urinoma.

4. Arteriovenous fistula may occur after penetrating injuries.

5. Pseudohydronephrosis due to accumulation of urine in the perinephric tissues.

6. Renovascular hypertension.

Clinical picture :

1. History of trauma

2. General :

- There may be hypovolemic shock.
- Picture of associated injuries.

3. Pain : Localized to renal area.

4. Microscopic or gross hematuria. Hematuria will be absent in :

1. If there is complete avulsion of the pedicle.
2. If there is a ureteral injury.
3. Sever hypotension.
4. Clot retention.
5. Tear not involving the pelvi-calyceal system.

5. Intestinal ileus is common in the presence of retroperitoneal bleeding.

6. Oliguria : due to hypotension or retention of urine due to clots in the bladder.

Signs :

1. Shock or signs of hemorrhage.

2. Hematuria : in collected urine samples.

3. Local tenderness. Guarding & rigidity of abdominal muscles with **rebound tenderness.**

4. A mass in the flank may be caused by extravasated blood & urine. If the overlying peritoneum has been torn, it will leak into the peritoneal cavity causing diffuse abdominal tenderness.

Investigations :

[A] Laboratory :

1. Hematocrete & urine examination for hematuria
2. Renal function tests.

[B] Radiological investigations :

1. Ultrasound or CT scan.

2. Plain film may reveal :

(a) Distortion of the renal shadow or obliteration of the psoas shadow.
(b) Scoliosis of the spine.
(c) Associated rib or spin fractures.

3. IVU : May show :

(a) Normal function and configuration if the injury is minimal.
(b) Deformed renal pelvis or calyces.
(c) Extravasation of dye.
(d) Non-visualization e.g. in total pedicle avulsion, arterial thrombosis or severe contusion.
(e) Confirm the presence of a functioning kidney on the opposite side.
(f) Displacement or deformity of the renal pelvis or ureter.

4. Renal isotope scanning (DMSA) : Can delineate main renal laceration and is useful in patients allergic to contrast material.

5. Angiography.

Treatment : Most renal injuries will be cured by conservative management.

{A} Conservative treatment :

1. Bed rest in a hospital (Return to work is allowed only one week after complete cessation of microscopic hematuria.)
2. Analgesics.
3. Large fluid intake.
4. Broad spectrum antibiotics.
5. Follow up chart for:
(a) Pulse, blood pressure.
(b) The size of any perirenal mass.
(c) Urine for the R.B.C.'s counts.
(d) Hemoglobin and hematocrete.

{B} Surgical treatment : Indicated for :

1. Failure of medical ttt indicated by persistent hematuria or failure of the vital signs to stabilize (**S**hock).
2. Presence of a progressively enlarging perirenal **S**welling.
3. Open injuries.
4. Presence of an **A**ssociated intraperitoneal injury.
5. Evidence of **P**erirenal infection or abscess.
6. Indications for delayed surgery:
(a) Hydronephrosis.
(b) If hypertension develops, vascular repair or nephrectomy is performed.

Principles of surgical repair :

1. Transperitoneal approach.
2. Exploration of the abdomen for possible associated injuries.
3. Preliminary control of the vascular pedicle using vascular clamps.
4. The collecting system is closed.
5. Defects of the cortical tissue are approximated by sutures.
6. If one pole of the kidney is avulsed , **partial nephrectomy** is done.
7. **Nephrectomy** is performed if the kidney is shattered or if there is complete avulsion of the vascular pedicle provided that the other kidney is functioning well.
8. Treatment of associated injuries.

Ureteric injuries

Etiology : Iatrogenic injuries are the most frequent.

1. Blunt or penetrating trauma is relatively rare.
2. Surgical injury during difficult or extensive gynecologic operations or abdominoperineal resection of the rectum.
3. Endoscopic urological procedures e.g. ureteroscopy.
4. After radiation therapy to the pelvis.

Pathology : The following consequences may take place.

[1] Extravasation of urine which may be :

(a) Intraperitoneal : Leading to peritonitis or urinary fistula.

(b) Extraperitoneal : A retroperitoneal collection can cause reflex paralytic ileus.

[2] Ureteric obstruction :

(a) Complete obstruction : causes suppression of urine production from the corresponding kidney.

(b) Partial obstruction : causes hydronephrosis.

[3] Anuria and acute renal failure may complicate bilateral injuries or those affecting the only functioning kidney.

Clinical features :

1. History of trauma or surgery.
2. Nausea, vomiting, loin pain and probably fever.
3. Oliguria or anuria in bilateral cases. Symptoms and signs of acute renal failure develop in neglected cases.
4. Urinary leakage or fistula.
5. Renal tenderness and/or renal mass (hydronephrosis).

Investigations :

1. Ultrasound
2. Intravenous pyelography
3. Ureteric catheterization and retrograde pyelography.

Complications :

1. Stricture formation with hydronephrosis.
2. Ureteric urinary fistula.
3. Renal failure.

Treatment :

Exploration of the ureter and repair of injury by end-to-end anastomosis or by reimplantation of the lower end of ureter into the bladder.

Traumatic rupture of the bladder

- Traumatic rupture of the bladder may be intra or extraperitoneal.

Intraperitoneal rupture bladder	Extraperitoneal rupture bladder
Etiology	
More in males.	More in males.
Bladder full and direct trauma applied to suprapubic region (either opened or closed injury).	Bladder is injured secondary to fracture pelvis e.g. run over accident (Bladder may not be full).
Instrumentation (cystoscopic procedures).	Instrumentation.
Pathology	
Urine escapes into peritoneal cavity and may lead to peritonitis.	Urine extravasates in the pelvic cellular tissue, then ascends up between peritoneum and transversalis fascia.
Symptoms	
Hypovolemic Shock +	Hypovolemic Shock ++
Suprapubic Pain +	Pain +
Urine retention **with no desire** to micturate.	Retention **with desire** to micturate.
Symptom of peritonitis.	Symptoms of toxaemia from cellulitis.
	Fracture pelvis.
Signs	
Suprapubic tenderness and rigidity.	Suprapubic tenderness and rigidity.
Later signs of peritonitis.	Swelling and edema of anterior abdominal wall.
Bladder can not be felt.	Bladder may be felt.
Catheter can be passed easily and **no urine is obtained.**	Catheter can be passed easily and **some urine is obtained.**
Complications	
1. Pelvic abscess from infection of a hematoma or urine collection. **2. Delayed peritonitis.** **3. Partial incontinence** if bladder neck is injured.	
Investigations	
Free.	**1. X-ray** May be fracture pelvis.
2. IVU Excludes other urinary injuries & lack of filling of the urinary bladder.	
3. Ascending cystogram Provides a definite diagnosis by demonstrating leakage of contrast outside the bladder.	
Treatment	
• Treatment of shock. • Abdomen is opened , urine in peritoneum is sucked. Tear in bladder closed and bladder drained by cystotomy tube. • Peritoneum is drained.	• Treatment of shock. • Suprapubic extraperitoneal exploration of bladder. • Tear in the bladder is closed and bladder is drained by cystostomy tube. • The pelvic fracture is then treated according to its type. It should be noted that internal fixation of the broken bone is contraindicated in the presence of urine extravasation, for fear of causing osteomyelitis.

Rupture of the urethra

Sites : The injury may affect :

1. The anterior part of the urethra = extra pelvic type.
2. Posterior urethra = pelvic type.

Types : The injury is either complete or incomplete.

Extra pelvic Rupture (Injury of Bulbous urethra)	Intra pelvic Rupture (Injury of membranous urethra)
Affects the bulbous urethra.	Affects membranous urethra near apex of prostate.
Etiology	
Fall astride a projecting object , kick to perineum or instrumentation.	Fracture pelvis which tears the puboprostatic ligament & displaces bladder & prostate upwards.
Clinical Picture	
Pain in perineum.	Pain and Hypovolemic shock.
Urethral bleeding. ++	Urethral bleeding. +
Inability to pass urine.	Inability to pass urine.
Bladder distended.	Bladder distended.
Perineal hematoma.	Picture of fracture pelvis.
NB : The triad of Urethral hemorrhage , Perineal hematoma and Retention of urine is diagnostic.	
Extravasation of urine	
To the perineum , scrotum , penis then to lower part of the anterior abdominal wall (anterior to abdominal muscles). • Urine can't pass downwards into the thigh because of attachment of membranous layer of superficial fascia to deep fascia.	In the pelvic cellular tissue (similar to extra peritoneal rupture bladder).
Complications	
1. Urine extravasation. 2. Stricture of urethra. 3. 2ry infection in neglected cases.	1. Urine extravasation. 2. Stricture of urethra. 3. 2ry infection in neglected cases. 4. Incontinence. 4. Impotence.
Investigations	
Negative.	**1. X-ray** — Fracture pelvis.
2. Ascending urethrogram Shows the site of extravasation.	
P/R	
Negative.	Prostate can't be felt being displaced upwards.
Treatment	
The patient is instructed not to void urine. • Passage of a catheter is contraindicated as it can increase the damage and introduce infection. • If there is no clinical extravasation , diagnosis is obtained by doing urgent ascending urethrogram. • **If the urethrogram shows extravasation suprapubic cystostomy is urgently performed.** After three weeks the urinary bladder is filled with a contrast material and if stricture develops , endoscopic urethrotomy followed by repeated dilatation are performed or surgery.	

Inflammation of the urinary tract

Acute pyelonephritis

Etiology :

[A] Routes of Infection :

1. Ascending infection : From lower urinary tract.
2. Hematogenous route (uncommon) : In cases of bacteremia e.g. in infective endocarditis.
3. Lymphatic route.

[B] Predisposing Factors :

1. Stagnation of urine due to obstruction at any level of urinary tract.
2. Vesico-ureteric reflux.
3. Impaired immunity.
4. Introduction of infection through catheterization , cystoscopy or bladder operations.

Clinical picture :

Symptoms :

1. Acute onset of fever with rigors , headache & malaise.
2. Acute pain in the loin radiating to the groin.
3. Frequency , urgency , dysuria & strangury may occur.
4. Nausea , vomiting & may be diarrhea.

Signs :

1. Fever , tachycardia , pallor & sweating.
2. Tenderness & rigidity over the affected kidney.
3. Signs of complications.

Complications :

1. Septicemia.
2. Perinephric abscess.
3. Pyonephrosis.
4. Acute renal failure (rare).
5. Recurrence of infection & progression to chronic pyelonephritis.

Investigations :

1. Urine analysis.
2. Urine culture & sensitivity test.
3. Renal function tests.
4. Blood picture.
5. Renal imaging.
6. Investigations to detect the cause.

Differential diagnosis :

Acute pyelonephritis should be differentiated from other conditions presenting with :

1. Fever with rigors.
2. Abdominal pain.
3. Pyuria e.g. cystitis.

Treatment :

1. Antimicrobial drugs & urinary antiseptics.
2. Change the urine pH
3. Symptomatic treatment : e.g. analgesics & antipyretics as aspirin.
4. Treatment of complications.
5. Correction of predisposing factors : e.g. calculi , obstruction.

Chronic pyelonephritis

Etiology :

It usually occurs in patients with underlying abnormalities e.g. obstruction , vesico-ureteric reflux & impaired immunity.

Clinical picture :

1. Recurrent attacks of loin pains , frequency , dysuria & fever.
2. Non-specific symptoms as fatigue , backache , abdominal discomfort anorexia & loss of weight.
3. Gradual progression to chronic renal failure.
4. Hypertension.
5. Anemia.
6. Tubular dysfunction e.g. salt-losing nephropathy , proteinuria.

Investigations : See before.

Treatment :

1. Correction of the predisposing factors.
2. Antibiotic therapy :
   - Selection of antibiotics is based on culture & sensitivity tests
   - Antibiotics are used in reduced doses because of impaired excretion of the drugs.
   - Prolonged & repeated courses of treatment may be needed.
3. Treatment of chronic renal failure & hypertension if present
4. Unilateral nephrectomy is rarely performed in advanced unilateral cases with complete loss of function.

Cystitis

Etiology :

Predisposing causes :

1. Instrumentation , calculi & foreign bodies.
2. Constant re-infection from the upper or lower urinary tract.
3. Lowered general resistance.

Routes of infection :

1. Descending from the kidney.
2. Ascending from the urethra , prostate and vulva or during instrumentation.
3. Lymphatic from the cervix uteri , prostate , seminal vesicles or bowel.
4. Direct through a vesicovaginal or vesicointestinal fistula.
5. Hematogenous from distant foci of infection , such as boils , carbuncles and infected tonsils.

Organism : E. coli is by far the commonest organism and may occur in pure or mixed culture.

Clinical picture :

Symptoms :

1. Increased frequency of micturition is the chief symptom.
2. Pain is felt in the suprapubic and perineal regions.
3. Hematuria.

Signs :

1. There is tenderness over the bladder on abdominal , rectal or vaginal examination.
2. In cases secondary to pyelonephritis , tenderness is elicited over one or other renal angle.

Investigations :

1. Urine analysis : A mid-stream specimen in male or a catheter specimen in female should be sent for complete bacteriological examination including culture & antibiotic sensitivity tests.

2. Radiography and pyelography.

3. Cystoscopy reveals the precise pathological changes , and local predisposing causes in the bladder , urethra or prostate.

Pyonephrosis

Definition & Etiology :

Pyonephrosis is a chronic retention of infected urine in the kidney. **It may be:**

1. Primary : due to coincident infection and obstruction.

2. Secondary : due to infection of a pre-existing hydronephrosis.

Pathology :

- The renal parenchyma is hollowed out forming multilocular cavities filled with pus and lined with necrotic tissue. There is marked perinephritis with dense adhesions.
- Secondary calculi, with the formation of a calculus pyonephrosis, are common.
- Like hydronephrosis, a pyonephrosis may be :
  - **1. Closed :** when the obstruction is complete.
  - **2. Open :** when the obstruction is incomplete.

Complicatios :

1. Septicemia & septic shock.
2. Renal failure.

Clinical features :

- The cardinal symptoms are **Pain , Swelling , Fever & Pyuria.**

1. Swelling : In primary types : small with restricted mobility.
In secondary types : large with more marked mobility.

2. Pyuria : May be absent when the pyonephrosis is closed and the bladder free from cystitis.

Investigations :

[A] Laboratory.

[B] Radiography :

1. A plain X-ray : may reveal the presence of a calculus.
2. IVU : shows delayed or absent secretion. It will assess the condition of the other kidney.
3. U.S.
4. Ascending pyelography : Required in many cases to :
   (a) Confirm the diagnosis.
   (b) Estimate the degree of dilatation.
   (c) Drain the kidney.

[C] Cystoscopy :

1. Shows signs of chronic cystitis and a purulent efflux from one ureteric orifice.
2. A ureteric catheter : May be arrested at the site of obstruction **OR**
   May enter the pelvis and gives exit to purulent urine with marked relief of pain.

D.D. :

1. Basal pneumonia & pleurisy.
2. Acute cholecystitis.
3. Acute appendicitis.
4. Acute pancreatitis.
5. Perinephric abscess.

Treatment :

[1] Infection of an obstructed kidney should be treated as an emergency.

Antibiotics are prescribed and the kidney is drained by **inserting a large percutaneous nephrostomy tube or ureteric catheter.** Any cause of obstruction as a stone or stricture should be corrected.

[2] Nephrectomy :

- In advanced cases, the ideal treatment is **primary nephrectomy** provided the opposite kidney is healthy.
- Rarely, when septic absorption has caused marked deterioration of the general condition, **preliminary nephrostomy** has to be carried out.

Renal carbuncle

The lesion attacks the cortex of the kidney, with much destruction of the parenchyma. The condition may remain undiagnosed for weeks or months. Sometimes, the lesion bursts causing a perinephric abscess.

Perinephric abscess

Etiology :

1. Primary perinephric abscess is rare. The infection is blood-borne.
2. Secondary perinephric abscess is much commoner. Due to extension of infection from the kidney, or neighbouring structure, such as a retrocecal appendix, the gall bladder, spine, pleura or pelvic organs.

Pathology :

Organisms : Staphylococci, streptococci.
The pus is situated between the kidney and its perinephric sheath and may extend downwards into the pelvis or may burst through the perirenal fascia to reach the loin.

Clinical features :

1. Pain, swelling & constitutional manifestations.
2. A gradually increasing swelling appears in the loin and extends backwards and outwards.
   - The swelling is ill-defined and does not move with respiration.
   - There is often slight flexion of the hip from spasm of the psoas muscle.

Investigations :

1. X-ray : Shows :
(a) Curvature of the spine (scoliosis) away from the affected side.
(b) Obliteration of the psoas shadow.
(c) Elevation and fixation of the diaphragm.
2. Ultrasound or CT scan can accurately diagnose the abscess.

Treatment :

[1] In early cases : Antibiotics and hot fomentations.
[2] In established cases : immediate drainage through an adequate lumbar incision. The kidney is palpated to detect any unruptured cortical abscess, which should be drained.

Renal tuberculosis

Incidence :

1. Age : Rare in children, usually in young adults (20 – 40 years of age).
2. Sex : males > females.
3. The disease is unilateral in the early stages, but may attack the opposite kidney.
4. Always secondary T.B.

Types :

1. Primary : The renal lesion is the only active focus in the urogenital tract.
2. Secondary : The infection is already present in one of the genital organs.

Etiology :

1. The primary form : due to hematogenous infection from a hidden focus in the body.
2. The secondary form : due to ascending infection along the ureter or periureteral lymphatics from the prostate, seminal vesicles or urinary bladder. The causative organism is human type of mycobacterium tuberculosis in 75% of cases and bovine strain in 25%.

Pathology :

[1] T.B. pyelonephritis :

- Infection starts at the apex of the pyramid with passage of T.B. bacilli in urine and no macroscopic change (Stage of sterile acid pyuria).
- Involvement of the pelvis leads to T.B. pyelonephritis.

[2] Open T.B.:

- Destruction of the kidney substance with cavitation leads to ulcerocavernous type of T.B.
- Infection spreads outside the kidney forming T.B. perinephric abscess.
- Infection spreading along the ureter (T.B. ureteritis with stricture formation) or infection may reach the bladder and prostate. Descending infection leads to T.B. infection of the vas and epididymis.

[3] Closed T.B.:

- In some cases the pelvis is obstructed and T.B. hydronephrosis or atrophy of the kidney (Auto-nephrectomy) develops.

Clinical features :

{I} In the early stages : Frequency of micturition is the chief symptom.
{II} In the later stages : 4 groups of symptoms are present :
[A] Vesical symptoms : There is progressive frequency of micturition. **Causes :**

1. Polyuria from inflammation of the kidney.
2. Irritation of the bladder by tuberculous products.
3. Tuberculous cystitis.
4. Contraction of the bladder from fibrosis.
5. Secondary infection.

> The frequency is usually associated with **suprapubic pain** & in late cases with **terminal hematuria (strangury).**

[B] Renal symptoms :

- Dull ache in the loin.
- The pain may occur on the sound side from compensatory hypertrophy.
- Passage of tuberculous debris or blood clots may give rise to ureteric colic.

[C] Changes in the urine :

1. Acidic sterile pyuria.
2. Hematuria may be present.

[D] General symptoms of T.B.

Clinical examination :

- Usually negative.
- Occasionally an enlarged tender kidney may be palpable.
- General examination and P/R & scrotal examination must be carried out to exclude other T.B. foci.

Investigations :

[1] Plain X-ray chest , Tuberculin Test , ESR and **PCR.**
[2] Examination of the urine : will show the tubercle bacilli detected by Zeihl-Neelsen staining, culture or animal inoculation. In closed lesions the bacilli fail to appear in the urine.
[3] Radiography :

1. A plain X-ray : may show calcification in the kidney or secondary calculi.
2. Intravenous urography : often reveals an irregular dilatation of a calyx with a characteristic **moth-eaten appearance** due to erosion of the apex of a pyramid.

[4] Cystoscopy :

1. Tubercles appear on and around the ureteric orifice, ulcers appear later.
2. In longstanding cases, gaping and retraction of the ureteric orifice **(golf-hole ureteric meatus).**
3. The capacity of the bladder is progressively diminishes **(thimble bladder).**

Treatment :

[A] Medical treatment : Anti T.B. treatment.

[B] Surgical treatment : Indicated mainly to treat the complications of the disease.

1. Renal cavernotomy : In case of localized pyocalyx, the best treatment is to :
(a) Expose this cavity.
(b) Remove its wall.
(c) Swab its wall until healthy granulation tissue is reached.

2. Pyeloplasty : for stricture at the pelviureteric junction.

3. Contracted bladder : is treated by ileocystoplasty (augmentation cystoplasty).

4. Nephroureterectomy : If one kidney is grossly affected provided that the other kidney is functioning.

Bilharziasis of the bladder

Incidence : The urinary bladder is the commonest site of bilharzial infection ***due to :***
1. The large venous channels which allow free passage of the parasite.
2. The strong muscle coat with its vigorous contractions drives the ova through the bladder wall.
3. The elastic mucosa is readily penetrated by the ova.
4. The urine acts as an amniotic fluid and guards against premature hatching of the ova.

Age : Between the ages of 10 and 30.
Sex : Male : Female = 4:1.

Etiology :

Schistosoma hematobium and only in 4% Schistosoma mansoni.

Pathology :

1. Terminal hematuria : Due to extrusion of the ova through the mucosa of the bladder.
2. Granulomatous lesions in the submucosa : Due to retained ova in the wall of the bladder.
3. Bilharzial pseudo-tubercles : Larger and more prominent than those of T.B.

Fusion of these pseudotubercles leads to :

4. Bilharzial nodules.
5. Granulomata (Bilharzial masses) : Due to an aggregation of nodules. They are sessile, soft and bleed on touch.
6. Papillomata : Like granulomata but they are pedunculated.

7. Sandy patches : Due to calcified dead ova with degeneration of the overlying epithelium.
8. Ulceration : Due to sloughing of mucous membrane containing dead ova.
OR : Due to sloughing of a bilharzial papilloma.

9. Fibrosis : Leading to decrease in the capacity of the bladder i.e. contracted bladder.
- Fibrosis of the lower end of ureter may lead to **hydronephrosis.**
- Fibrosis of the bladder neck leads to **bladder neck contracture.**

10. Carcinoma : As an end result complication. It is usually squamous cell carcinoma.

Complications :

1. Secondary infection (very common).
2. Stone formation.
3. Ureteric obstruction.
4. Bladder-neck obstruction. It may result from :
(a) Papillomata.
(b) Ring fibrosis around the internal meatus.
(c) Bilharziasis of the seminal vesicles and prostate.
5. Pericystitis : leading to the formation of a dense fibrous sheath, which causes progressive contraction of the bladder.
6. Malignancy.

Clinical features :

The earliest symptom of bilharzial infection is **the swimmer's or bather's itch**, which may be followed after 4 - 12 weeks by fever, urticaria and asthma, but such symptoms usually pass unnoticed until the bladder is invaded by ova usually within 6 months after penetration of the cercariae.

1. Hematuria is the cardinal symptom. It is usually slight and terminal.
2. Difficulty in micturition : At first there may be a slight difficulty. In late cases, severe difficulty may occur from bladder neck obstruction, stone formation or bilharzial ulceration.
3. Pain : In the early stages, little pain may be felt in the urethra. But, with the development of ulceration, stone formation and malignancy, the pain is markedly increased.
4. Frequency : From congestion of the trigone, secondary cystitis and other complications.

Investigations :

1. Urine examination : In the acute stage, the urine contains living bilharzial ova.
2. Cystoscopy and Cystography.
3. Plain radiography : May reveal bilharzial calcification of the bladder, or calculi.
4. IVU : may show strictures of ureters, hydronephrosis or bladder neck obstruction.

Treatment :

[A] Medical treatment : Antibilharzial drugs.
- **Urinary antiseptics** should be given if secondary cystitis is present.

[B] Surgical treatment : Depends on the individual lesion :

1. Bilharzial papillomata :
(a) Rarely precancerous and require no special treatment.
(b) If indicated for removal :
i. Cystoscopic fulguration : if they are few.
ii. Partial cystectomy : if bulky and suspicious of malignancy.
2. Ulcers : usually respond to conservative treatment.
In resistant cases, cystoscopic diathermy coagulation or partial cystectomy is employed.
3. Bladder neck obstruction : needs correction by surgery or by per urethral resection.
4. Lower ureteric stricture : may require urteroscopic ballon dilatation or uretro-vesical implantation.

Tumors of the urinary tract
Neoplasms of the Kidney

1-benign: adenoma, angioma, and angiomyolipoma.

2. Malignant

Most of the neoplasms of the kidney are malignant.

[1] Tumors arising from the renal parenchyma (90%) : These include renal cell carcinoma, nephroblastoma and some rare tumors as leiomyosarcoma, hemangiopericytoma and angiomyolipoma.

[2] Tumors arising from the renal pelvis (10%) : These includes two types :

(a) Transitional cell carcinoma.
(b) Squamous cell carcinoma.

Renal Cell Carcinoma (Hypernephroma, Grawitz's tumor)

This is the commonest renal parenchymal tumor representing 75% of all renal neoplasms.

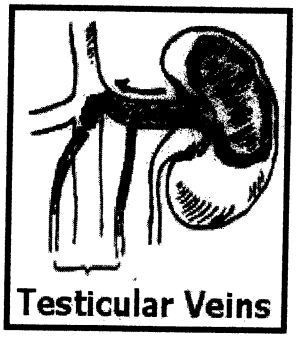
Incidence :

1. Sex : The male to female ratio is 2:1.
2. Age : The majority of patients are between 50 & 70 years old.
3. Side : Bilateral tumors are either synchronous or metasynchronous & occur in 1-2% of cases.

Etiology :

1. Using tobacco products.
2. Von-Hipple Lindau disease : in which there may be renal and pancreatic cysts, cerebellar hemangioblastomas, retinal angiomata and pheochromocytoma.
3. Loss of the short arm of chromosome 3 is a constant chromosomal change.

Pathology :



Gross appearance :

- The tumor usually starts in one pole of the kidney (usually the upper).
- The color of the tumor is golden yellow due to its high lipid content.
- Areas of hemorrhage and necrosis.
- There is an apparent pseudocapsule surrounding the lesion.
- The tumor infiltrates the renal pelvis early in the course of the disease.

Microscopic appearance :

- Adenocarcinoma that arises from the cells of the proximal convoluted tubules.

Types :

1. Usually clear cell pattern as cholesterol crystals in cytoplasm dissolve during preparation.
2. Granular cell type is due to the increased mitochondria in the cytoplasm.

Spread :

[1] Direct spread : The neoplasm soon infiltrates the renal pelvis. Then the Zukercandle's **(Gerota's)** fascia and may even infiltrate adjacent structures.

[2] Lymphatic spread : To the lymph nodes along the hilum of the kidney, then to the para-aortic lymph nodes and finally to the thoracic duct.
[3] Blood spread : Hematogenous spread gives rise to metastases in the lungs ***(cannon-ball secondary deposits)***, bones, and brain. Sometimes, a malignant thrombus may be present in the renal vein or extends even to the IVC.

Clinical picture :

[1] Hematuria : (in 50% of cases).
It is described as being painless, recurrent, profuse and total hematuria. Sometimes, the blood passes as tubular clots taking the shape of the ureter.

- **Any patient with hematuria should be investigated for the possibility of neoplasms of the urinary tract.**

[2] Pain : (In 40% of cases). It may be due to :
(a) Stretch of the renal capsule.
(b) Passage of blood clots causing ureteric colic.
(c) Infiltration of the adjacent lumbar nerves.

[3] Renal mass : (In 30% of cases).
An irregular hard, renal swelling.

- **The classical triad of hematuria, pain and renal mass is present in only 10% of patients and they indicate an advanced disease.**

[4] Metastases : May be the first presentation e.g. pulmonary or skull deposits.

[5] Non specific symptoms : as fever, night sweats, weight loss or anemia.

[6] Secondary varicocele : A rapidly enlarging varicocele which doesn't empty by elevation of scrotum.

[7] Systemic syndromes :
(a) Hypercalcemia : occurs in 5% of cases due to secretion of a parathormone-like substance by the tumor or due to the presence of bone metastases.
(b) Erythrocytosis.
(c) Amyloidosis.

Investigations :

1. Plain X-ray (KUB) : may show mottled calcification.
2. IVU may reveal :
(a) Enlargement of the kidney.
(b) Elongation, displacement, compression or amputation of a calyx.
(c) Displacement of the renal pelvis.
3. Renal sonography : can distinguish solid from cystic masses.
4. CT scan is very accurate :
(a) It can determine the extent of local spread.
(b) It can detect lymph node enlargement and invasion of the renal vein or inferior vena cava.
5. Angiography : It has now been replaced by CT scan
6. Chest X-ray & Isotope bone scan : for metastases.

Staging :

Robson staging system for renal cell carcinoma :
Stage I :
Tumor is confined to the kidney.
Stage II :
Tumor is confined to Gerota's (Zukercandle's) fascia and involves the perinephric fat.
Stage III :
Tumor involves the renal vein and/or regional lymph nodes.
Stage IV :
Distant metastases (lungs, liver or bones).

Treatment :

[A] Operable patients : (stages I & II)

Radical nephrectomy is performed. It entails removal of the kidney within its Gerota's fascia together with the ipsilateral adrenal gland.

Ligate the vascular pedicle as the first step of the operation for 2 reasons :

1. Prevention of dissemination of malignant cells during manipulation of the tumor.
2. To be able to remove a malignant thrombus from the inferior vena cava, if present.

- Patients who have hypernephroma in a solitary kidney are treated by **partial nephrectomy** removing the lesion with 1 - 2cm safety margin.

[B] Inoperable patients :

Patients who have metastatic disease are treated by **Irradiation, Alpha or Gamma Interferon** and **Interleukin-2.**

Wilm's tumor (Nephroblastoma)

This tumor arises from embryonic nephrogenic tissue & contains epithelial & connective tissue elements.

Incidence : This neoplasm represents 10% of childhood malignant tumors :

- 90% of cases occur before 7 years of age.
- Peak incidence is 3 to 4 years of age.
- Male to female ratio is equal.

Pathology :

Gross picture :

- The tumor appears as a solitary sharply demarcated, apparently encapsulated mass with hemorrhage and necrotic areas.
- Renal pelvic invasion is rare.
- Renal vein invasion may occur.

Microscopic picture : The tumor contains both :

1. Epithelial cells may form primitive glomeruli or tubules.
2. Connective tissue elements as areas of cartilage, fat, smooth or striated muscles are present.

- Degrees of differentiation of the tumor vary from :
  Favourable histology (FH) to **Unfavourable histology (UH).**

Anomalies associated with Wilm's tumor :

1. Hemihypertrophy : Asymmetrical growth of one side of the body in 3% of cases.
2. Genito-urinary anomalies : in 4% of cases as renal hypoplasia, hypospadias or cryptorchidism.
3. Aniridia : in 2% of cases.
4. Increase in the incidence of neurofibromatosis.

Clinical picture :

1. The main presentation (90%) is **an abdominal mass** which is smooth, firm and is confined to one side of the abdomen.
2. One third of patients present with **vague abdominal pain.**
3. Microscopic hematuria occurs in 50% of cases.
4. Hypertension is present in up to 60% of cases. It results from encroachment on the blood supply producing renal ischemia and excess renin production.

D.D. :

1. Neuroblastoma : irregular tumor, hard in consistency & may cross the midline.
2. Hydronephrosis. 3. Multicystic kidney. 4. Infantile type of polycystic kidney.

Investigations :

[A] Laboratory :

1. Complete blood count, liver and renal functions.
2. Urine catecholamines help to rule out neuroblastoma.

[B] Radiologic:

1. IVU : Reveals an enlarged kidney, the pelvi-calyceal system is distorted, the kidney may be non-functioning. It will assess the function of the other kidney.

2. Ultrasonography :

(a) Can differentiate solid from cystic renal masses.
(b) Can detect liver metastases.

3. CT scanning :

(a) It can detect involvement of surrounding structures or involvement of contralateral kidney.
(b) It can follow the tumour response to chemo and radiotherapy.

4. Chest X-ray : to detect pulmonary metastases.

National Wilm's tumor staging system :

This is a **post-nephrectomy staging** on which further management will depend.

Stage I : Tumor is limited to the kidney.
Stage II : Tumor extends beyond the kidney but is completely excised.
Stage III : Residual non hematogenous tumour confined to the abdomen.
Stage IV : Hematogenous metastases.
Stage V : Bilateral renal involvement at diagnosis.

Treatment :

- If possible, the affected kidney is radically resected as for renal cell carcinoma.
- Unresectable lesions should be treated with chemotherapy and are re-explored; usually the tumor can then be removed.
- Remaining tumor in nodes or other organs should be marked with surgical clips to facilitate direction of radiation therapy.

Scheme for post-operative management :

1. If there is no residual focal lesion : adjuvant chemotherapy using actinomycin D, vincristin & adriamycin.

2. If there is residual focal lesion : combination of radiotherapy & chemotherapy is applied.

Prognosis: chemo and radiotherapy have improved the overall prognosis to 80% 5-year survival. Early cases are usually cured.

Tumors of the renal pelvis

[A] Transitional Cell Carcinoma (TCC) (Urothelial Tumor) :

Tumors of the renal pelvis, ureter or bladder which are transitional cell carcinoma are called urothelial tumours.

TCC of the renal pelvis accounts for 7% of all renal tumours. TCC spreads with implantation so patients with TCC of the renal pelvis have a 30% chance of developing the same lesion in the bladder and a 2% chance of developing a contralateral tumour.

Clinical picture :

Gross or microscopic hematuria is present in 90% of patients.

Treatment :

The classic therapy is nephroureterectomy with excision of a bladder cuff.

[B] Squamous Cell Carcinoma of the Renal Pelvis :

- Surface implantation does not occur.
- Often associated with infection and stone.

Tumors of the Urinary bladder

Benign Papilloma

Pedunculated tumor, often less than 1 cm in diameter.

Carcinoma of the Urinary Bladder

The main predisposing factors in Egypt are Schistosomiasis and cigarette smoking.

Classification:

55%: Transitional cell carcinoma.
40%: Squamous cell carcinoma.
5% : Others including adenocarcinoma.

Squamous Cell Carcinoma	Transitional Cell Carcinoma
Etiology	
This usually occurs on top of long standing bilharzial cystitis which is usually complicated by secondary infection. Many theories are suggested to explain the possible role of bilharzial cystitis as a predisposing factor : [1] Continuous **I**rritation by the ova as they pass through the bladder wall. [2] Chronic **I**nfection especially by ammonia producing organisms may lead to squamous metaplasia. [3] Abnormalities of **T**ryptophan metabolism leading to the production of carcinogenic compounds. [4] Increased level of B-**G**lucuronidase enzyme which transforms non carcinogenic compounds to a carcinogenic compound. [5] **N**.**N**itroso compounds. Nitrates are excreted in urine where they are acted upon by bacteria leading to the liberation of N.Nitroso compounds which are carcinogenic.	[1] **C**arcinogens : industrial carcinogens : (a) Aniline dyes. (b) Rubber and textile industries due to exposure to aromatic amines. [2] **C**igarette smoking. [3] **C**yclophosphamide. [4] **P**elvic irradiation. [5] **P**rolonged intake of large doses of phenacetin. [6] **A**rtificial sweeteners, e.g. saccharin, (not well established).
Pathology	
Preneoplastic alterations of the urothelium : **1. Brunn's nests :** are clusters of cells situated in the submucosa. **2. Cystitis cystica :** is Brunn's nest that has undergone central liquefaction. **3. Cystitis glandularis :** the transitional cell lining of the cysts has changed to columnar epithelium. **4. Squamous metaplasia.** **5. Vesical leukoplakia :** is cornification of a normally non cornifying membrane.	

Site : Commonest in the posterior and lateral wall and rarely in the trigone.

Gross appearance :

The tumor may present as :

1. Nodular fungating type (commonest).
2. Malignant ulcer.
3. Papillary tumor (Rare).

Microscopic appearance :

(See sq. C.C. of the skin)

The tumor is composed of keratinized cells & usually contains concentric aggregates of cells (squamous pearls).

Squamous cell cancers shed keratinized cells in urine that can be detected cytologically in most patients.

Site : Common sites are the base, trigone and around the ureteric orifices.

Types :

1. Localized erythematous lesions :
Represent foci of carcinoma in situ (CIS).

2. Papillary tumors :
Have a structure that is similar to the benign papilloma, but the epithelial lining shows malignant characters.

3. Nodular tumors :
Have the appearance of a raised plaque with a broad base. The surface is nodular. Microscopically the cells are arranged as solid sheets enclosed by bands of vascular connective tissue.

4. Ulcerative type :
Shows as a malignant ulcer.

According to the degree of cellular differentiation, TCC is classified into :

Grade I : Well differentiated.
Grade II : Moderately differentiated.
Grade III : Poorly differentiated.

According to depth of invasion of the bladder wall :

1. Superficial TCC : The lesion is usually papillary.
2. Muscle invasion TCC : These are usually solid. There is invasion of the muscle layer.

Spread

Delayed : due to :
Fibrosis & calcification in the bladder, perivesical tissues & local L.N.'s.
The incidence of lymph node metastasis is 15%.
The incidence of distant metastasis is 4%.

Early: as there is no calcification or fibrosis.

[A] Direct spread :

From the mucosa to muscle coat to perivesical tissue then to surrounding structures :

1. Upwards : Peritoneum, bowel and omentum.
2. Downwards : Prostate in males.
3. Laterally : Lateral pelvic wall, sacral plexus and pelvic fascia around the rectum.
4. Posteriorly : In male, to seminal vesicles and rectovesical pouch. While in females, to vagina and uterus.

[B] Lymphatic spread : From Perivesical L.N's. to external iliac & internal iliac L.N's. then to common iliac L.N's. & para-aortic L.N's.

[C] Blood spread : Late.

Staging

Wallace classification for bladder cancer is based on bimanual clinical examination as follows : **T0 :** No palpable mass. **T1 :** Palpable mobile mass, but no induration of the bladder wall. **T2 :** Palpable mass, with induration of the bladder wall. **T3 :** Palpable mobile wall with extravesical spread. **T4 :** Fixed bladder mass.	P0: No tumor in the specimen. Pis : Carcinoma in situ. Pa : Tumor confined to the mucosa. P1 : Tumor confined to the submucosa. P2 : Superficial muscle invasion. P3a: Deep muscle invasion. P3b: Perivesical fat invasion. P4a: Invasion of near by organ, e.g. prostate. P4b: Fixed to pelvis or abdominal wall.

Clinical Picture

Type of patient

Farmer with history of bilharziasis. Male to female = **4:1.** **Peak age incidence :** 20 - 50 years.	**No history of bilharziasis.** Male to female = **3:1.** **Age :** usually above 60 years.

Symptoms

1. The patient usually presents by recent exaggeration of the **symptoms of cystitis** which have been present for a long time. > There is **burning micturition, frequency & pyuria.** **2.** There may be recurrent attacks of **painful hematuria.** **3. Necroturia :** means the passage of pieces of whitish tissue representing necrotic tumor. **4.** There may be **suprapubic pain.** **5.** Rarely, the tumor may cause **urinary retention.** **6. Symptoms due to spread** e.g. sciatica.	• **Hematuria :** is painless. • **Cystitis :** is later due to secondary infection. **Otherwise the clinical picture is the same.**

Signs

- **General examination :**
  The patient may have anaemia or signs of renal failure.

- **Per rectal and bimanual examination :**
  Assess the size, site and mobility of the tumor.

- **Abdominal examination:**
  Mass : hydronephrotic kidney or hepatomegaly may be felt.
  In case of bilharziasis : splenomegaly may be present.

Investigations

[A] Laboratory tests :

1. Urine examination : for R.B.C's and pus cells.
2. Cytology: for malignant cells.
3. Blood urea and creatinine.
4. Liver function tests.

[B] Imaging :

1. Plain x-ray : may show eaten calcification of the urinary bladder in cases of bladder carcinoma on top of bilharzial cystitis.
2. Intravenous urography : will reveal the presence of the lesion (as irregular filling defect) and will assess the renal function.
3. Abdominal & pelvic ultrasound : can asses the renal size, the bladder mass & liver metastases.
4. CT scan : can assess the extent of the tumor and any pelvic node deposits.
5. Cystoscopy and biopsy are mandatory :
In case of T.C.C. transurethral resection of visible tumor with biopsy of the underlying bladder muscles is done for proper staging. Random biopsies are also taken to diagnose carcinoma insitu.

Treatment

Unfortunately in Egypt by the time the diagnosis of carcinoma of the urinary bladder is made, the case is too advanced.

Attempts to cure these patients by :
1. Transurethral resection (TUR). OR
2. Partial cystectomy
are not feasible.

Squamous cell carcinoma is also resistant to chemo or radiotherapy, and so the best, chance for the patient is to do **A Radical Cystectomy operation.**

This procedure entails resection of :
In males : the whole bladder with its perivesical fat, the prostate, the seminal vesicles and the lower ends of both ureters.
In female : the urinary bladder, the uterus, the tubes and the anterior vaginal wall are removed.

Block dissection of the internal and external iliac and obturator lymph nodes is performed in both sexes.
Urinary diversion is then performed.

Prognosis :
The 5- year survival following this procedure is 32%.

[A] Superficial bladder cancer (Pis Pa, P1) :

1. Transurethral resection (TUR) :
Excising the tumor with the underlying muscle plus multiple random biopsies to detect carcinoma in situ.
2. Laser ablation :
Using Nd YAG laser.
3. Intravesical chemotherapy (adriamycin) or immunotherapy (BCG vaccine) :
To prevent recurrence.
It is indicated in grade II and III tumors and in the presence of carcinoma in situ.

Regular follow up for 5 years is indicated to detect recurrence. If there are multiple recurrences or persistent carcinoma in situ, the patient is treated by **Radical Cystectomy.**

[B] Muscle invasive TCC :
This is treated by either :
1. Radical cystectomy : This operation differs from that described for SCC in only one step ; the urethra has to be removed because of the tendency of TCC to spread along the urethra.

2. Radiotherapy : The results are less successful than surgery.

3. Chemotherapy : For metastatic disease or when the surgical margins are not free. Best combination is **"MVAC".** Methotrexate, Vinblastine, Adriamycin and Cis-platin.

Calcular disease & obstructive uropathy

Urinary calculi (General)

Incidence :

- Urinary calcular disease is a common disorder affecting 10-20% of the population at some stage of their life.
- Males are affected more than females.
- Calculi occur mainly in middle aged persons but no age is excluded.

Pathology :

I- Etiology :

[1] Geography : increased incidence in mountainous, desert, tropical areas & Mediterranean countries.

[2] Climate and seasonal factors : There is a relationship between higher environment temperature and increased incidence of urinary stone disease.

[3] Water intake : Both the volume of water ingested and the mineral or trace element content of the water supply are important factors in the aetiology of stone disease.

[4] Diet :

(A) Some food stuffs are rich in certain crystals e.g.

i. Spinach, tomatoes, berries and mango are rich in oxalates.
ii. Liver, kidneys, sardines, Salmon & ducks are rich in purines which form uric acid crystals.
iii. Milk and its products are rich in calcium.

(B) Vitamin A deficiency.

(c) In civilised countries in which the diet is nutritious, calculi occur mainly in the kidneys whereas in poor countries, bladder calculi are common.

[5] Metabolic and endocrinal factors : These vary according to the type of the stone.

(A) Ca oxalate and Ca phosphate calculi :

i. Hypercalcemia due to hyperparathyroidism.
ii. Hypercalciuria (most frequent cause).
iii. Hyperoxaluria :
  (a) Primary hyperoxaluria: This is a rare congenital illness
  (b) Acquired hyperoxaluria: This is seen with enteric diseases
  (c) Idiopathic hyperoxaluria: Occurs with eating foods containing high amounts of oxalate.
iv. Hypocitraturia is present in 10% of patients with recurrent calcium oxalate calculi.
v. Hyperurcosuria is present in 20% of patients with recurrent calcium oxalate calculi.
vi. Renal tubular acidosis is usually associated with hypercalcuria and hypocitraturia.

(B) Uric acid calculi :

i. Hyperuricemia e.g. in gout.
ii. Hyperurcosuria.

(C) Cystine calculi are due to hereditary cystinuria.

(D) Xanthine calculi are due to hereditary xanthinuria.

[6] Infection :

Stones associated with chronic urinary tract infection are called Struvite or triple phosphate stones. These stones are caused by urea splitting bacteria as Proteus, Pseudomonas, and Klebsiella, but not E. coli. These organisms alkalinize the urine and produce increased concentrations of bicarbonate, phosphate, and ammonium ions.

[7] Congenital anomalies : All congenital anomalies of the urinary tract predispose to stasis, infection and stone formation.

II- Theories of stone formation :

- More than one of these mechanisms is involved :

1. Nucleation : Stone formation is initiated by the presence of a crystal or foreign body in urine already supersaturated with a crystallizing salt.
2. Matrix initiation : An organic matrix of serum & urinary proteins provides a framework for deposition of crystals.
3. Absence of inhibitor : Absence or low concentration of normal urinary stone inhibitors such as mucoproteins, citrate, pyrophosphate & magnesium permits crystallization.
4. Epitaxy : Certain substances such as calcium oxalate & uric acid have a similar crystal structure of lattice. This allows overgrowth of one crystal on the surface of another.

III- Initial sites of stone formation :

1. Randall's plaques : Deposition of calcium on the basement membrane of collecting tubules & on the surface of papillae.
2. Carr's concretions : Calcium salts deposited in the renal lymphatics produce obstruction and separate lymphatics from the collecting tubules.
3. Intranephronic calculosis : Intratubular deposits of calcific cellular debris or organisms.

IV- Types of urinary calculi :

Incidence :

1. Calcium Oxalate > 70%
2. Calcium phosphate > 70%.
3. Struvite (Triple phosphate) > 15%.
4. Uric acid > 7-9%
5. Cystine & Xanthine > Very rare

[1] Calcium oxalate calculi : The most common type.
(a) Calcium oxalate monohydrate calculi : Smooth, dark, hard stones with a dense radio-opaque shadow. Less often they have irregular mammillary processes, the so called "mulberry stone".
(b) Calcium oxalate dihydrate calculi : Lighter in color, with a granular surface, less radio-opaque, and much easier to fragment.
(c) The "jackstone" : Is a mixed stone combining both hydrates with a central mass and radiating specules.
[2] Calcium phosphate calculi : A smooth, yellowish white and radio-opaque. They grow in alkaline medium and they can attain a large size, e.g. the staghorn calculus that fills the renal pelvis and calyces.
[3] Triple phosphate (Struvite) calculi : Occur in alkaline urine and grow very rapidly, often filling the renal pelvis and calyces. They are friable, smooth, yellowish white and they are radio-opaque. They are formed of ammonium, magnesium and Calcium phosphate.
[4] Uric acid calculi : Hard, smooth and golden yellow. Pure uric acid calculi are radiolucent.

Symptoms of calcular disease of the urinary tract

Symptoms of renal and ureteric calculi :

[1] Pain :

(A) Renal Stone :
A stone fixed in the renal pelvis will cause fixed renal pain located in the renal angle posteriorly, in the hypochondria anteriorly or in both areas. The pain is increased by movements.

(B) Ureteric Stone :
i. A stone in the upper third : Will cause colicky abdominal pain in the loin which is referred to the groin & the testicles in males or labia majora in females.
ii. As the stone moves to the middle third : The pain is felt in the iliac region and may simulate appendicitis.

iii. A stone impacted in the lower end : Will cause frequency of micturition and there will be pain at the end of micturition referred to the tip of the penis and often associated with strangury.

- **During an attack of renal pain or ureteric colic, there is usually nausea or vomiting.**

[2] Hematuria : During an attack of ureteric colic, the urine is usually smoky due to microscopic hematuria. Gross hematuria is uncommon but may occur.

[3] Pyuria : Calcular disease predisposes to urinary tract infection & microscopic or gross pyuria may be present.

Physical signs :

1. Fever is rarely present unless there is urinary tract infection.
2. Examination of the abdomen reveals moderate deep tenderness over the site of the stone.
3. There is often **tenderness over the posterior flank** as well.

Urinary Bladder Stones

Etiology :

[A] In children : Secondary to dehydration, low protein diet or secondary to urinary tract infection.
[B] In adults : the common causes are :
1. Urinary obstruction, e.g. urethral stricture or benign prostatic hyperplasia.
2. Presence of urinary tract infection.
3. Neurogenic bladder and sphincteric dysfunction.
4. Foreign body as an indwelling catheter.

Types :

1. Primary calculi develop in aseptic urine as calcium oxalate and uric acid calculi.
2. Secondary calculi which occur in the presence of infection as Struvite stones.

Clinical features :

1. Pain : Due to contraction of the bladder around the stone at the end of micturition.
Site : In the suprapubic area and is referred to the tip of the penis. It is more frequent at day time and is aggravated by movements.
2. Frequency of micturition : Due to irritation of the bladder mucosa especially the trigone.

- At first the frequency is only during daytime, but if cystitis develops, it will be nocturnal and diurnal.

3. Difficulty in micturition : Interruption of the stream. Acute retention may develop.
4. Hematuria : May occur at the end of micturition.

Urethral Stones

Origin :

1. Most of them have migrated from the ureter and become arrested in the prostatic, bulbous or penile urethra.
2. Less commonly calculi occur primarily in the urethra in a diverticulum or above a stricture. These primary calculi are composed mainly of calcium phosphate.

Clinical picture :

- During micturition there is **severe urethral pain, interruption of the stream** followed by **retention of urine.**
- Primary calculi usually do not cause too much symptoms
- Examination can usually detect the stone.
- If a metal bougie is passed along the urethra, a characteristic click is felt.
- **Plain X-ray is diagnostic.**

Investigations for urinary calculi

Urine analysis :

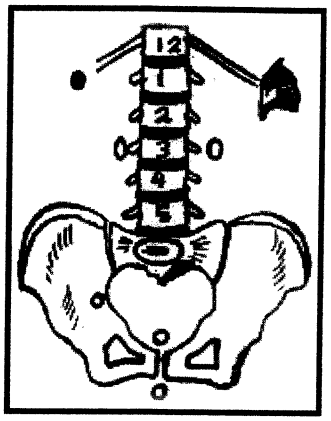
1. There is microscopic or gross hematuria.
2. Pus cells.
3. Crystals of the same type that are creating the calculus.

Imaging :

[A] Plain Abdominal Film (KUB) :

In most patients, the position and outlines of the kidneys can be seen especially after relief of the colic. The course of the ureter is characteristically parallel to the lateral processes of the lumbar spine; the ureter then crosses the pelvic brim and turns laterally into the true pelvis before again turning medially to enter the bladder.

D.D. of radio-opaque shadows on radiography :



1. Calcified lymph nodes : usually multiple, Mottled.
2. Gall stones : multiple, faceted, with signet ring appearance.
   > A lateral view will reveal them anterior to the bodies of vertebrae.
3. Foreign bodies in the gastrointestinal tract.
4. Phleboliths : circular shadows in the pelvis.
5. Calcified tip of the 12th rib.
6. Hyperossified areas in bone.
7. Chip fracture of transverse process.
8. Calcified lesions in the kidney, e.g. tuberculosis. Calcified suprarenal glands.

[B] IVU :

1. Shows obstruction by the stone.
2. Gives an idea about renal function bilaterally.
3. Useful in radiolucent calculi which appear as filling defects.

[C] Ultrasonography :

1. Visualize the kidney and the degree of obstruction.
2. Helpful in radiolucent calculi

> Disadvantages : It is difficult to view almost all of the ureter.

Occasionally the following investigations may be needed :

Radioisotope Methods : Usually reserved for patients who are sensitive to contrast material.

Ascending (Retrograde) Pyelography : Reserved for radiolucent calculi difficult to be localized by other techniques.

Management

I- Management of the patient during an attack of pain :

1. Hospitalization may be necessary.
2. Parenteral analgesics (opiates) may be required.
3. Antispasmodics & anti-inflammatory agents in particular aminophylline & indomethacin.
4. Adequate hydration.
5. Antibiotics if there is suspicion of urinary tract infection.
6. It is important to recover any stone or gravel that is passed by filtering the urine or micturating in a clean container. Stone analysis allows planning of future therapy.
7. Decide the line of treatment.

II- Management of urinary calculi :

The aim of treatment is to remove the present calculi and to eliminate the possibility of recurrent stone formation.

[A] Conservative treatment :

Indications :

1. Stones less than 5mm in diameter.
2. No evidence of back pressure effect or infection.

Method :

1. The main item is high fluid intake.
2. During an attack of pain the previously mentioned measures will be applied.

[B] Intervention treatment for urinary tract calculi :

Renal stones

Indications for interference to remove renal calculi :

1. A stone more than 5mm.
2. Growing stones.
3. Evidence of obstruction or secondary infection.
4. Persistent pain.
5. Gross hematuria.

Methods of removal of renal calculi :

1. Extracorporeal shock wave lithotripsy (ESWL).
2. Percutaneous nephrolithotomy (PCNL).
3. Combined PCNL and ESWL.
4. Open surgery.
5. Chemolysis of stones.

[A] Extracorporeal shock wave lithotripsy (ESWL) :

Indications : All stones less than 2cm whether calyceal or pelvic.

Contraindications :

[A] Urological contraindications :

1. Associated pathology.
2. Acute episode of renal infection.
3. Distal obstruction.
4. Large stone.
5. Renal insufficiency.
6. Stones of the lower calyx.
7. Solitary kidney except with stenting of ureter (to avoid acute obstruction by the ureteric stone fragments which may cause acute renal failure).

[B] Non urological contraindications :

1. Pregnancy.
2. Bleeding diathesis.
3. Bone abnormalities like kyphosis or marked scoliosis (location of stone is very difficult).

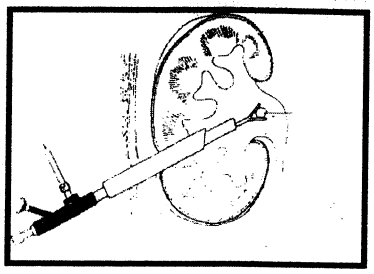
Advantages of ESWL :

1. Painless, non invasive technique which can be done as an outpatient procedure.
2. Suitable for risky patients.
3. Successful for most urinary calculi except the hard and radiolucent.

Complications (very minimal) :

1. Transient attacks of hematuria.
2. Colicky ureteric pain.
3. Failure to disintegrate the stone.
4. Fever due to acute obstruction by the fragments.

[B] Percutaneous Nephrolithotomy PCNL :



The procedure involves two steps :

1. Access to the pelvis of the kidney : This is achieved by placing a guide-wire through the flank under fluoroscopic or ultrasound control. After placement of guide-wire, the tract is dilated to a size sufficient to accommodate the nephroscope with a sheath 24-30 F.

2. Stone removal : The stone is directly extracted through the sheath. If it is large, disintegration of the stone can be achieved by ultrasonic, laser, compressed air or electrohydraulic lithotripsy.

Indications of PCNL :

1. It is nowadays confined to those situations where ESWL is contraindicated or failed.
2. As a combined therapy in big or staghorn calculi. Debulking of the stone by PCNL is performed at first, followed by complete disintegration of the remaining part by ESWL.

Contraindications for PCNL :

1. Bleeding diathesis.
2. Relative contraindications :
   (a) Congenital anomalies. (b) During pregnancy. (c) Marked scoliosis or kyphosis.

Advantages :

1. Small endoscopic wound. 2. Mild post-operative pain. 3. Short hospital stay.

Complications of PCNL :

1. Rarely injury may occur to the colon, duodenum, spleen or pleura.
2. Complications related to stone removal :
   (a)Bleeding. (b) Failure to get the stone. (c) Residual stone.

[C] Open surgery :

It is indicated only for those patients in whom there is a contraindication to ESWL or PCNL or if these measures failed.

The possible operations include :

1. Pyelolithotomy : This means opening the renal pelvis and removal of the stone.

2. Nephrolithotomy : This means incision through the renal parenchyma.

3. Pyelonephrolithotomy : Combines the above mentioned two procedures. It is indicated for branched calculi.

4. Lower partial nephrectomy : Indicated for a stone impacted in the lower calyx with narrowing of its neck.

5. Nephrectomy : If the kidney is non-functioning, provided that the contralateral kidney is functioning.

Ureteric stones

Indications to remove ureteric stones :

Urgent indications :

1. Acute complete obstruction of a solitary kidney or bilateral obstruction.
2. Evidence of secondary infection.

Less urgent indications :

1. Stone larger than 8mm.
2. Persistent pain.
3. Failure of conservative treatment.

Procedures :

[I] Upper third of the ureter :

1. If the stone is less than 1cm, it is pushed up to the kidney by a ureteric catheter and the ESWL is used.
2. If the stone is more than 1cm, open surgery is required (through lumbar incision).

[II] Middle third of the ureter : Open surgery is recommended. Ureteroscopic extraction at this site is risky, and ESWL is very difficult (through Abernathy's incision).

[III] Lower third of the ureter :

1. If the stone is less than 1cm : either :
   (a) Disintegrated by direct contract lithotripsy, ultrasonic waves, laser or electrohydraulic shock waves.
   (b) Extract the stone by the dormia basket forceps.
2. If the stone is larger than 1cm, open surgery is required.

Stones in the lower 2/3 of ureter above ischial spine are exposed through Abernathy's incision.
Stones in the lower end of ureter below ischial spine are exposed through a midline suprapubic incision.

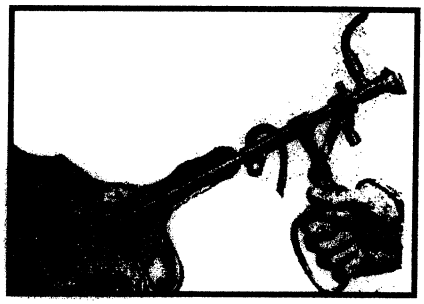
Stones of the urinary bladder

Stone less than 2 cm :

Can be crushed mechanically by the lithotrite (litholapaxy) or can be disintegrated by ultrasonic waves or electrohydraulic shock waves. The fragments are then lavaged outside the bladder.

Indications for open surgery (cystolithotomy) :

1. If the stone is larger than 2cm.
2. Multiple stones.
3. Very hard stones.
4. In children.
5. Failure of crushing or disintegration.
6. Presence of another pathology needing surgery e.g. diverticulum.



Urethral calculi

[A] Prostatic urethra :

A metal bougie is passed along the urethra in an attempt to dislodge the calculus in the bladder where it is crushed by litholapaxy and the fragments evacuated.
If this procedure fails, the calculus is removed by open surgery through an incision in the bladder.

[B] Membranous and bulbous urethra :

One of the following methods is used :

1. Removal of the stone by a urethral forceps passed through a urethroscope.
2. A number of filiform bougies are passed beyond the stone, twisted and then pulled out together holding the stone.
3. If the previous measures fail, open surgery is performed.

Metabolic work-up to prevent stone recurrence :

[A] The stone should be chemically analyzed.

[B] The following investigations should be performed :

1. Serum calcium and phosphorus to exclude hyperparathyroidism.
2. 24 hours urine collection for : Calcium - Uric acid - Oxalates – Citrates

[C] Advice to be given to patients with urinary calculi :

1. High fluid intake especially in hot weather.
2. Certain precautions according to the type of the stone, directed mainly to avoid certain diets (see before) & treating any metabolic disorders if possible (e.g. Gout).

Obstructive uropathy

Etiology :

- **According to the level of obstruction :**

[1] Urethra :

1. Congenital : Meatal stenosis & Posterior urethral valves.
2. Stone.
3. Strictured urethra : Traumatic.
Inflammatory.
Neoplastic.

[2] Prostate :

1. BPH.
2. Carcinoma of the prostate.

[3] Bladder neck obstruction :

1. Congenital : Marion's disease.
2. Acquired : Bilharzial cystitis.

[4] Urinary Bladder :

1. Tumors involving bladder neck or uretro-vesical orifices.
2. Neurogenic bladder.
3. Stones.
4. TB & Bilharziasis.

[5] Ureter :

(a) In the lumen : Calculi.
(b) In the wall :
1. Congenital : Atresia.
2. Traumatic stricture : following trauma or surgery.
3. Inflammatory stricture : following TB or Bilharziasis.
4. Papilloma or carcinoma of the ureter.
5. Functional : vesico-ureteric reflux.

(c) Compression of the ureter from outside :
1. Infiltration by carcinoma of cervix , colon or rectum.
2. Idiopathic retro-peritoneal fibrosis.
3. Aberrant vessels.

[6] Causes at the Pelvi-ureteric junction and Intra-renal :

1. Idiopathic Pelvi-ureteric Junction Obstruction **(PUJO).**
2. Stricture due to TB.
3. Stones.

Pathological Changes : {Back pressure changes above the obstruction}

[1] Urethra : Urethral dilatation proximal to the obstruction.

[2] Urinary Bladder :

- **Early in the compensated phase :** Hypertrophy of the muscular layer of the baldder. The wall of the bladder becomes trabeculated and diverticulae may occur.
- **Later :** The bladder wall thins & The bladder dilates.

[3] Ureter :

Early : Muscular hypertrophy of the wall.
Later : Atony of the wall of the ureter occurs. The ureter becomes dilated, elongated & tortuous **(hydroureter).**

[4] Kidney :

- **The pressure within the kidney** is normally close to zero. With obstruction or reflux , this pressure will increase and the pelvi-calyceal system will dilate leading to hydronephrosis.
- **The higher the obstruction** the greater is the effect upon the kidney.
- **First :** The normal concave appearance of the calyces becomes flat then rounded.
- **The parenchyma** becomes compressed between the renal capsule and the increasing intra-pelvic pressure.
- **At a later stage ,** the kidney is completely destroyed and appears as a thin-walled sac filled with clear fluid or with pus.
- **The fluid in the renal pelvis** is constantly changing by reabsorption followed by secretion.
- **As unilateral hydronephrosis progresses ,** the normal kidney undergoes compensatory hypertrophy of its nephrons.

Investigations :

1. **Blood urea & creatinine.**
2. **US :** will reveal :

(i) Size of the kidney.
(ii) Any distention of the pelvi-calyceal system.
(iii) Thickness of the renal cortex.

3. **Plain X-Ray UT & IVU :** If creatinine is normal.
4. **Retro-grade Cystography or Pyelography :** If IVU is not conclusive.
5. **Cystoscopy & Urethroscopy :** to detect any abnormality in the urethra or the bladder.
6. **Cystometry & Uroflowmetry :** to diagnose neurogenic bladder.
7. **Radionuclide study.**

Complications :

1. **Infection :** leading to 2ry pyonephrosis.
2. **Stone formation.**
3. **Renal insufficiency :** in bilateral cases.
4. **Rupture of the kidney.**

Aims of Treatment :

1. **ttt of the cause.**
2. **Relief of obstruction.**
3. **Eradication of infection.**

NB: If one kidney is completely destroyed and the other kidney is normal, nephrectomy is indicated.

Factors affecting morphological and functional changes following obstruction:

1-The level of obstruction: affecting one or both kidneys
2- Degree of obstruction.
3- Duration of obstruction.
4- Development of infection.

Acute Obstructive Problems in The Urinary Tract

1. Acute retention of urine.
2. Calcular anuria.
3. Accidental ligation of both ureters during a surgical procedure.

Calcular Anuria

This is a serious problem in which there is anuria due to calcular obstruction in the ureter.

Etiology :

1. A stone obstructing the ureter of the only functioning kidney ; the other kidney being non-functioning , surgically removed or congenitally absent.
2. Calculi obstructing both ureters.

Clinical Picture :

- Usually there is a **long history** of calcular disease.

[A] Stage of onset :

The patient may experience an **attack of ureteric colic** followed by **anuria**, but the history of ureteric colic may be absent.

[B] Stage of tolerance :

Colic disappears & there is no major symptoms or signs. Still there is no urine output. Urea & creatinine start to rise.

[C] Stage of uremia (Renal Failure) :

If the patient is not treated, the **clinical picture of uremia** will occur within few days.

How to determine the site of obstruction ?

The answer is not always easy ; but the usual site of obstruction is :

1. The site of recent **ureteric or renal pain.**
2. The site on which there is **renal tenderness or guarding.**

D.D. :

1. Other causes of anuria.
2. Retention of urine

Investigations :

[1] Insert a urinary catheter : to check that the bladder is empty.
[2] Plain X-Ray : Not very helpful ; as the stone is small & the patient is distended due to uremia.
[3] Abdominal US (very useful) : will reveal distended pelvi-calyceal system on the affected site. It should be remembered that a large hydronephrotic kidney is not usually the site of obstruction as it is often non-functioning.

Treatment (Urological Emergency) :

Drainage of the obstructed kidney :

1. A cystoscope is passed and **a ureteral catheter** is passed along the affected ureter beyond the stone. If the site of obstruction is not exactly known , both ureters should be catheterized.
2. If ureteric catheterization fails , a percutaneous nephrostomy is performed.

- **Later , the patient is properly investigated and the stone is extracted.**

> **NB :** Following the relief of urinary obstruction , the patient may have severe diuresis where he may pass several litres of urine per day. The fluid loss should be replaced.

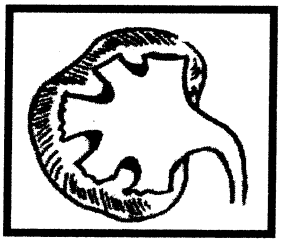
Hydronephrosis

This is chronic aseptic distention of the renal pelvis and calyces due to partial or intermittent obstruction of the urinary tract. It may be Unilateral or Bilateral.

Etiology : See Before (Obstructive Uropathy)

Pathological Changes : See Before (Obstructive Uropathy)

Pathology : There are two types:

Pelvic type	**Renal type**
The pelvis of the kidney is of the extra-renal type.	The pelvis of the kidney is of the intra-renal type.
In advanced cases , the pelvis forms one large chamber with the kidney capped over part of it.	In advanced cases , the kidney becomes transformed into a lobulated sac with rounded bosses.
This occurs when the obstruction is **high.**	This occurs when the obstruction is **low.**
The pelvis dilates and bulges forwards. The pelvi-ureteric junction is raised and kinked.	The calyces gradually become dilated , with progressive atrophy of the renal cortex.
	

Clinical Picture :

1. **Pain :** There is usually slight dull aching pain or a sense of discomfort in the loin.
2. **Swelling :** In late stages.
3. **Urine :** There is often polyuria of low specific gravity.
4. **Picture of the cause.**
5. **Manifestations of renal failure,** if present.

Complications : See Before (Obstructive Uropathy)

D.D.: Of palpable hydronephrosis :

1. Polycystic kidney.
2. Hypernephroma.

Investigations : See Before (Obstructive Uropathy)

IVU : will show :

1. Dilatation of the renal pelvis which will be biconvex.
2. Delayed excretion. in advanced cases , no secretion occurs.

Treatment :

1. Removal of the cause of obstruction is usually followed by recovery of the kidney.
2. In advanced cases of pelvic hydronephrosis , a plastic operation is performed to reduce the size of the pelvis and to provide adequate drainage of the lowest part of the pelvis.
3. If the kidney is non-functioning & the opposite kidney is normal , nephrectomy is recommended.

Benign Prostatic Hyperplasia (B.P.H.)

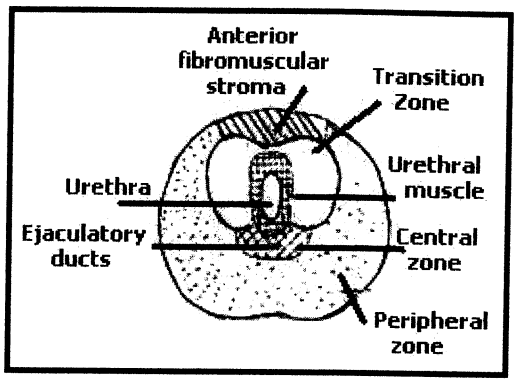
Incidence :

Benign prostatic hyperplasia is a condition affecting nearly 50% of males above the age of 50.

Etiology :

The most accepted theory is that B.P.H. is under endocrine control, with an unknown hormonal imbalance stimulating the transition zone prostatic cells to undergo hyperplasia.

Pathology :



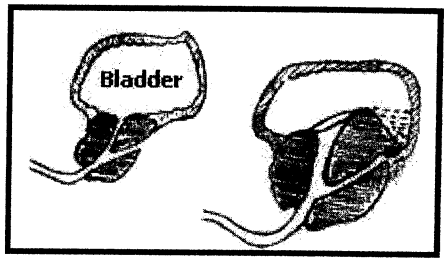
- The prostate is not divided into lobes.

Actually the prostate is divided into zones that are morphologically and functionally different :

1. Anterior fibromuscular stroma.
2. Peripheral zone.
3. Central zone.
4. Transition Zone (Periurethral area).

It is the transition zone which is the origin of B.P.H., with development of nodules that enlarge, compress the urethra and the rest of the prostate. The remaining compressed prostatic tissue forms the surgical capsule.

Microscopic picture :



- Hyperplasia is characteristically nodular.

Five microscopic appearances are identified :

1. Stromal. 2. Fibromuscular.
3. Muscular. 4. Fibroadenomatous.
5. Fibroadenomyomatous (most common).

Pathophysiology :

- On developing B.P.H. the large nodules may differentiate into lateral and middle lobes that obstruct both urethra & bladder neck.

The urethra becomes :

1. Elongated.
2. Narrowed as it is compressed laterally.
3. The normal posterior curve of the urethra becomes exaggerated.

The urinary bladder, ureters and kidneys : see before (chronic obstructive uropathy).

Clinical picture :

- There is no relation between the size of the prostate and the degree of symptoms.
- The symptoms depend upon the degree of obstruction of the urethra and the bladder neck by the enlarged gland.

[1] Frequency of micturition :

- Usually the earliest complaint.
- At first, the frequency is mainly nocturnal later the frequency occurs at day and night.

Causes of frequency :

1. Elongation of the posterior urethra and exposure of this sensitive mucosa to the urine.
2. Stretch of the internal sphincter allows few drops of urine to escape in the posterior urethra leading to **urgency.**
3. Later residual urine starts to accumulate in the bladder
4. Complications as cystitis, diverticulum or stone of urinary bladder.

[2] Difficulty in micturition :

(A) Difficulty to start :

- The patient has to wait for few seconds to start micturition **(hesitancy).**
- Strain will lead to congestion of the prostate with more obstruction and the patient learns that he has to relax.

(B) Difficulty to maintain : The stream is not forceful with diminution of its calibre.

(C) Difficulty to terminate : At the end of micturition there is inability to terminate micturition abruptly with post-voiding dribbling.

[3] Acute retention of urine :

- May be the first presentation.
- Precipitated by cystitis, constipation, cold weather, alcoholic drinks & sexual excitement.

[4] Chronic retention with overflow :

As the obstruction progresses, the bladder will be overdistended & may contain up to one litre of urine. The condition is painless & the actual complaint of the patient is incontinence.

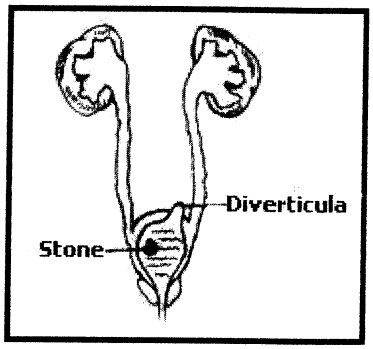
[5] Hematuria : May be severe. **Etiology :**

1. Rupture of a prostatic veins. 2. Another pathology e.g. bladder cancer.

[6] Sexual symptoms : At first the patient has an increased libido. Later, impotence may occur.

[7] Complications :

1. Bladder stone 2. Cystitis.
3. Bladder diverticulae 4. Chronic renal failure.



Physical examination :

General examination :

For evidence of chronic renal insufficiency.

Abdominal Examination :

May reveal a suprapubic mass (Full bladder). The kidneys may be enlarged.

P.R. Examination :

- The prostate is felt enlarged, smooth and firm.
- The median sulcus is preserved and the rectal mucosa can be moved over the prostate.

Complication : See before

Investigations :

I- Laboratory :

1. Routine laboratory tests : urine analysis, CBC, blood urea and creatinine and electrolytes.

2. Serum P.S.A. (Prostatic specific antigen) :

> Is to diagnose associated carcinoma of the prostate.
> The normal value is 0-4 ng/ml

II- Radiological :

[A] KUB : may show bladder or prostatic stones.

[B] Intravenous urography : The following is seen in B.P.H. :

1. Smooth basal filling defect in the bladder. There may be sacculation of the wall of the bladder or actual diverticulae.
2. Large amount of residual urine.
3. Hydronephrotic changes of the kidneys.
4. Hooking of ureters on entry into bladder (fish-hook sign).

[C] TRUS (Trans-rectal -ultrasound) :
1. To show non-palpable malignancy in the peripheral zone.
2. To assess the size of enlargement.
3. Ultrasonically guided biopsy may be taken.

[D] Abdominal sonar :
1. Measure the amount of post voiding residual urine.
2. Visualize the kidneys and the urinary bladder.

III- Instrumental :

Cystourethroscopy : Done in patients presenting with hematuria to exclude bladder pathology.

IV- Flowmetry :

A maximum flow-rate less than 15 ml/sec. indicates obstruction.

Treatment :

I- Conservative treatment :

Since the natural history of B.P.H. is not uniform, patients with minimal symptoms and no complications may remain as such for years. These patients may be placed on :

[1] Watchful waiting :
- Close follow-up & advice to avoid wetness, wine & withholding micturition for long periods.

[2] Medical treatment and watchful waiting :

(a) α-Blockers (Trasocine and Prasocine) : Improve symptoms, particularly frequency.

(b) 5-α-reductase inhibitors (proscar) :
- It improves patients' symptoms and actually diminishes the size of the enlarged prostate.
- In patients who improve on Proscar, the drug is to be continued for life.

II- Minimally invasive procedures :

- The aim of these techniques is temporary treatment when definitive treatment is contraindicated in high risk patient.

1. Thermotherapy : microwave heat therapy is applied by special probes per urethra or per rectum.
2. Endoscopic transuretheral cryo-ablation of the prostate.
3. Endoscopic transuretheral prostatic stents.

III- Surgical treatment :

Indications :

1. Distressing frequency.
2. Difficulty in micturition :
3. Acute retention of urine (If more than one attack).
4. Chronic retention with residual urine more than 200ml.
   - **Residual urine can be estimated by sonar or by post micturition cystography.**
5. Hematuria.
6. Presence of a complication as cystitis, diverticulae, stone or back pressure effects on the upper urinary tract.

The idea of surgery : Is to remove the adenomatous enlargement from inside the false capsule of compressed prostatic tissue.

Methods :

[A] Endoscopic surgery. **[B] Open surgery.**

[A] Endoscopic Surgery :

1. Trans-urethral resection of the prostate (TURP) : Using the cysto-resectoscope.
The prostate is removed piece by piece. It is the operation of choice for the majority of patients.
Contraindications : Large adenomas making the procedure lengthy and hazardous.

2. Visual laser Ablation of the prostate (V.L.A.P.) : Using the cystoscope.
Advantages : Bloodless procedure with post-operative results similar to T.U.R.
Drawback : the adenoma is not totally removed, with higher incidence of recurrence.

3. Transurethral heat prostatic vaporization : Using high energy electric heating.

[B] Open Surgery :

1. Transvesical prostatectomy.
2. Retropubic (Millin's) prostatectomy.

Indication :

1. Large adenomas (unfit for T.U.R).
2. Bladder pathology that needs surgery at the same time, e.g. a diverticulum or a big stone.
3. Mainly middle lobe enlargement.

Complications of surgery :

1. Bleeding (main problem) : This may be reactionary or secondary. Clot retention may occur.
2. Incontinence : occurs in one every 10.000 cases.
3. TURP syndrome : This is dilutional hyponatremia that rarely follows transurethral resection as a result of over absorption of irrigating fluid.
4. Retrograde ejaculation : due to damage of the internal sphincter.
5. Impotence : may occur in 2-5% of patients due to injury of pudendal nerve fibres in the region of the posterior urethra.
6. Infection : leading to urethritis, cystitis or epidydmo-orchitis.
7. Urethral stricture.

Prostate cancer

Incidence :

Carcinoma of the prostate is a common malignant lesion affecting males over the age of 65 years.

Predisposing factors :

1. Family history : The risk is increased if one or more first-degree relatives has the disease.
2. Diet : Dietary fat (not proven).
3. Race : African Americans have the highest incidence of the disease.
4. Age : Incidence shows constant rise over successive decades starting at the age of 50 years.

Pathology :

Gross picture :

1. The majority of neoplasms (75%) arise from the peripheral zone, beneath the capsule.
2. The tumor appears as hard infiltrating pale nodule.

Microscopic picture :

95% of prostatic cancer are adenocarcinoma derived from the acinar epithelium, 5% include transitional cell carcinoma or sarcoma.

Spread :

1. Direct invasion : involves the seminal vesicles, periprostatic tissue and rarely the rectum as the fascia of Denonvillier acts as a barrier.

2. Lymphatic spread : To the internal, external and common iliac lymph nodes then to the paraaortic lymph nodes.

3. Blood : 90% of blood borne metastases occurs in bones especially the lower vertebrae, pelvic bones and the ribs. This is due to the reversal of blood flow from the vesico-prostatic plexus to the emissary veins of the pelvic bones during coughing or sneezing. The metastases are usually osteoblastic. Rarely metastases develop in the lungs or the liver.

Clinical presentations : There are different presentations of the disease. Usually there are no symptoms or signs until late in the disease.

1. Pathological surprise :

After routine histopathological examination of the prostate removed with a diagnosis of BPH.

2. Urinary outflow obstruction :

- The patient may complain of **difficult micturition, hematuria** or **painful micturition.**
- A short history of these symptoms in a male above 65 years of age is suggestive.

3. Metastases :

The first presentation may be due to metastases. Backache, sciatica, spontaneous fracture or paraplegia .

4. Routine screening with prostate specific antigen (PSA) :

A high level raises suspicion & necessitates further investigations for confirmation of diagnosis

Bimanual rectal examination : Reveals a hard, irregular induration in a part or the whole of the gland. The median sulcus is obliterated. The rectal mucosa is not mobile.

- **It is to be noticed that a normal digital rectal examination does not exclude the diagnosis.**

Staging system TNM :

T0 : No clinical abnormality.
T1 : Nodule in one lobe.
T2 : Diffuse disease.
T3 : Extension to the seminal vesicles.
T4 : Lesion is fixed to other tissues.

N0 : No evidence of involvement of L.N.
N1 : Involvement of one regional node.
N2 : Involvement of several regional nodes.
N3 : Fixed regional nodes.
N4 : Involvement of common iliac or para-aortic nodes.

M0 : No evidence of distant metastases.
M1 : Distant metastases.

Investigations :

I- Laboratory :

1. Prostatic specific antigen (PSA) : It is the most useful marker in the diagnosis & follow up. It may be raised in non malignant conditions as in prostatitis or benign prostatic hyperplasia.

2. Acid phosphatase is raised. After rectal examination it remains elevated for 24 hours.

3. Alkaline phosphatase is raised if there are bony or hepatic metastases.

4. Blood picture and blood urea and creatinine.

II- Radiological investigations :

1. **Plain X-ray of the chest, pelvis & spine** to detect any metastases which are usually osteoblastic.
2. **IVU.**
3. **Bone scan.**
4. **CT scan of the pelvis.**
5. **Trans-rectal ultrasonography (TRUS) :** can give an accurate idea about the extent of the lesion.

III- Biopsy :

Any patient with abnormal digital rectal examination and/or an elevated PSA must undergo transrectal ultrasonography (TRUS) and 6 random biopsies are taken.

Treatment :

[1] Radical prostatectomy :

Indications : (a) Early localized disease. (b) Fit patient.
It entails removal of the prostate, seminal vesicles together with block dissection of obturator, internal & external iliac lymph nodes. The bladder is then anastomosed to the urethra.

[2] Irradiation :

Indications : (a) Locally advanced disease. (b) Unfit patients.
3. Symptomatic metastases : as it produces dramatic relief of pain.

[3] Transurethral resection :

Indications : for patients who have urinary outflow obstruction and are not fit for major surgery.

[4] Hormonal therapy :

Indication : This is the main line of ttt in advanced cases or in patients unfit for surgery or radiotherapy.
Method : Prostatic cancer is an androgen sensitive tumor. There are different methods for androgen ablation.
1. **Bilateral orchiectomy :** which means removal of testicular tissue from inside the tunica albuginea.
2. **Luteinizing hormone releasing hormone (LHRH) agonist (Zoladex).** The drug causes initial rise of the level of testosterone for 4-6 weeks then a drop to castrate levels. It gives equal results to castration and it has the same side effects. It is an expensive drug.
3. **Antiandrogens (Nutilamide) :** They are used as adjuvants to castration or LHRH agonists.
4. **Stilbestrol :** A small dose 1 mg t.d.s is used. It predisposes to deep vein thrombosis.

[5] No treatment in cancer prostate in small well differentiated tumors in ealderly men and may be manged by **watchful waiting** particulary if their life expectancy is less than 10 years.

Prognosis of prostatic carcinoma :

- The life expectancy with an incidental finding of focal carcinoma of the prostate is that of the normal population.
- With tumors localized to the prostate > The 10-years survival rate is **50%.**
- If metastases are present > The 10-years survival rate drops to **10%.**

Treatment of prostatic carcinoma

Early	Late
Radical prostatectomy.	**TURP.**
Radiation.	**Hormonal therapy.**
Watchful waiting.	

Urinary Bladder Diverticulae

A bladder diverticulum is a flask-shaped protrusion through the muscle wall of the urinary bladder.

Etiology :

1. Congenital diverticulum : Rare, solitary and located medial to and above the ureteric orifice and has muscle fibres within its wall (full thickness pouch) and is contractile.

2. Acquired diverticulae : Often multiple. They are always associated with distal obstruction, trabeculation and sacculation of the bladder. There are no muscle fibres in the fundus and they do not empty during micturition.

Complications :

Stagnation of urine in the diverticulum may result in :

1. Infection.
2. Stone formation.
3. Squamous metaplasia.
4. Rarely, a squamous cell carcinoma.

Clinical features :

- There are no specific symptoms to indicate the presence of a diverticulum.
- Diverticulae may fill with urine during micturition and subsequently empty into the bladder produce **an immediate desire to pass urine a second time (double micturition).**
- Complicated diverticulae will rise to **frequency, pain and hematuria.**

Diagnosis :

1. Cystography : Descending or ascending.
2. Cystoscopy : readily demonstrates diverticular openings within the bladder.

Treatment : (treatment of distal obstruction is mandatory)

Diverticulae are not excised unless complicated or producing ureteric obstruction.

Urethral stricture

Etiology :

1. Congenital : This occurs due to posterior urethral valves, meatal stenosis and may be associated with hypospadias.

2. Inflammatory :

(a) Following sexually transmitted diseases especially gonorrhoea which is the commonest inflammatory cause.
(b) Bilharziasis.
(c) Rarely tuberculosis.

3. Traumatic (the most common cause) :

The cause is either rupture of bulbous urethra in falling astride injuries or rupture of the membranous urethra with fracture of the pelvis. Another important cause of traumatic stricture is iatrogenic injuries due to indwelling urethral catheters, transurethral surgical or diagnostic procedures. Post-operative urethral stricture may occur following prostatectomy or amputation of the penis.

4. Neoplastic :

Urethral Polyps - Venereal warts - Carcinoma of the urethra.

Clinical picture :

1. Difficulty in micturition : The patient has to strain to pass urine. The stream is thin and weak. There is terminal dribbling of urine trickling form the dilated urethra.
2. Occasional passage of urethral discharge composed mainly of desquamated epithelium.
3. Frequency of micturition : due to incomplete evacuation of the bladder or due to cystitis.
4. Retention of urine. This may occur as acute or chronic retention. Acute retention may be precipitated by alcoholic excess or by activation of local infection.
5. In advanced cases all **the ill effects of obstructive uropathy** mentioned before will occur.

Complications :

1. Extravasation of urine with perineal or scrotal abscess formation.
2. Periurethral fistulae & abscesses.
3. Infertility.
4. Prostatitis.
5. Squamous cell carcinoma.

Investigations :

1. Uroflowmetry : reveals obstructed flow.
2. Urethrography : can localize the site, length and shape of the stricture.
3. Urethro-cystoscopy.
4. Ultrasonography.
5. IVU : to asses the renal condition.

Treatment :

[1] Urethral dilation :

- Under absolute sterile conditions and after installation of a local anaesthetic in the urethra.
- Dilatation is performed very gently.
- Dilatation usually starts with gum elastic bougies or filiform bougies in very narrow stricture.
- Metal bougies can be used later.
- If a filiform bougie can't be passed on three successful occasions, the stricture is considered to be impassable.

[2] Endoscopic visual internal urethrotomy :

- Through the urethroscope and under direct vision, the stricture is incised with sharp knife blade usually at the 12 O'clock position.

[3] Urethroplasty :

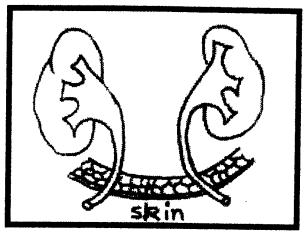
- This procedure is applicable for stricture of the bulbar and penile urethra if the previous measures fail.
- The idea is to excise the fibrous tissue of the stricture and then construct a new urethra using skin flaps from the penis, scrotum or perineum.
- For strictures following rupture of the membranous urethra , **Badenoch's pull through operation is indicated.**
- The idea is: Mobilize a long segment of the healthy anterior urethra and then pull up through the prostatic urethra.

Urinary diversion

Indications : Cystectomy, ectopia vesica, irremovable obstruction, loss of sphincteric control or in curable vesico-vaginal fistula.

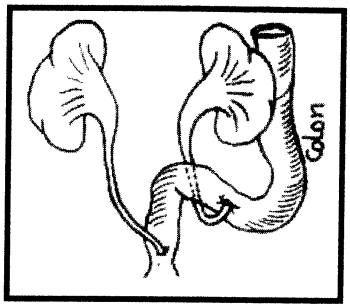
Urinary diversion following cystectomy : Many methods are available; the choice depends upon the condition of the patient and the surgical experience.

[A] Uretero-cutaneous implantation :



This operation is rarely performed as it has the following **Disadvantages :**
1. Stomal stenosis.
2. Sloughing of a portion of the ureter proximal to the stoma.
3. Continuous leakage of urine with excoriation of the skin.
4. Ascending infection.

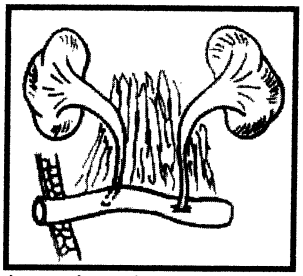
[B] Uretero-colic anastomosis :



- The two ureters are anastomosed to the sigmoid colon.

Advantages : The operation is simple, rapid and the patient is continent.
Disadvantages :
1. Recurrent upper urinary tract infections.
2. Deterioration of renal functions.
3. Hyperchloremic acidosis : as chlorides in urine can be absorbed by the colonic mucosa. To minimize this complication, the intake of sodium chloride is restricted and constipation is avoided.
4. Hypokalemia as intestinal secretions are rich in K (Secreted in excess due to irritation by urine).
5. Encephalopathy. Urea in urine is acted upon by the intestinal flora leading to the formation of ammonia. If the liver function is compromised, encephalopathy may occur.
6. Carcinoma of the colon in 30% of patients after a latent period of 10 years.

[C] Ileal conduit :



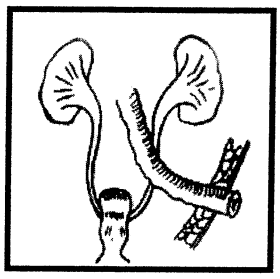
- A segment of terminal ileum is isolated with intact blood supply.
- The two ureters are implanted in the ileal segment, its distal end is closed and its proximal end is fixed to the skin as an ileostomy.
- A special appliance is applied to the ileostomy to collect urine.

Advantages : 1. No urine leakage. 2. No ascending infection.
Disadvantage: Hyperchloremic acidosis may occur but is less severe than with the colon diversion.

[D] Colonic conduit :

Based on the same idea as ileal conduit, but using a segment of sigmoid colon.

[E] Rectal bladder :



The sigmoid colon is divided above the recto-sigmoid junction; the proximal end is brought out as a colostomy. The distal end is closed and the two ureters are implanted in the rectum which will now work as a urinary bladder. **Advantages :**
1. Easy and safe operation.
2. The patient will usually be continent for urine.
3. No ascending infection or hyperchloremic acidosis.

[F] Continent urinary diversion :

- The idea is to separate a segment of bowel with intact blood supply, change its configuration so as to make it have a bigger volume and anastomose this segment to bladder neck or the remnant of urethra.
- Nowadays, this is the standard urinary diversion after cystectomy as it preserves renal function and is most physiological.

Orthopedics

Orthopedics

Orthopedics : Can be divided into traumatology (fractures and dislocation) and bone, joint disease and deformities.

Traumatology

Definitions :

1	**Fractures**	Is loss of continuity of bone.
2	**Dislocation**	Is loss of contact between the articular surfaces.
3	**Sublaxation**	Is a mild degree of dislocation with no wide separation of the articular surfaces.

Classifications of fractures :

[A] Fractures are either simple or compound :

Compound fracture : Is a fracture where the fracture haematoma is connected to the atmosphere with risk of infection.

- If, at operation, the laceration proves not to communicate with the fractur, the latter may be termed **{ technically compound }.**

- **Compound fracture may be :**

1. Compound from without : i.e. the causative trauma leads to skin wound & fracture.
2. Compound from within : i.e. the fractured end of bone is the cause of skin wound.
3. A break in the skin surface may be due to skin death from ischemia.

[B] The fracture may be simple or comminuted :

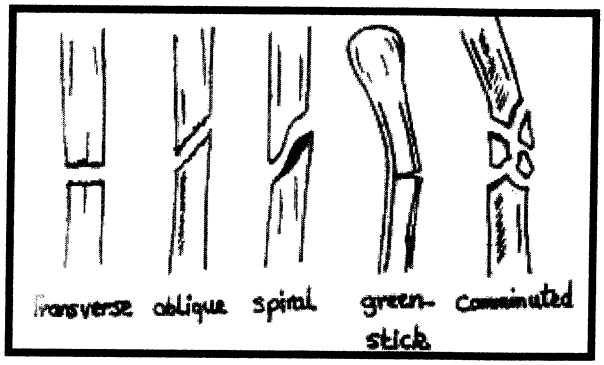
Comminuted : There are more than 2 pieces of bone at the fracture hematoma.

[C] The fracture may be complete or incomplete :

Incomplete as : Fissure fracture , greenstick fracture in which the cortex is broken from one side.

[D] Shape of the Fracture may be :

1. **Oblique.**
2. **Transverse.**
3. **Longitudinal.**
4. **Spiral fracture.**
   - All these shapes occur in longitudinal bones : e.g. femur, humerus.
5. **Depressed fracture in the skull.**
6. **Impaction.**



Causes of fractures :

1. **Direct trauma.**
2. **Indirect trauma :** e.g. fall on the out-stretched hand or fall from a height.
3. **Avulsion fracture :** e.g. fracture of the patella or the olecranon.
4. **Pathological fracture** : There is history of trauma not sufficient to cause fracture.

Phases of fracture healing:

1. Repair by granulation tissue.
2. Union by primary callus.
3. Formation of mature bone.

Causes of pathological fractures		
1	**Congenital**	1. Osteitis fragilis : The bone is fragile due to the deficiency in the osteoid tissue, while its mineral content is normal. 2. Congenital cysts.
2	**Inflammatory**	1. Chronic osteomyelitis. 2. T.B. osteomyelitis. 3. Gumma in the bone.
3	**Endocrinal**	1. Hyperparathyroidism leading to osteitis fibrosa cystica. 2. Cushing syndrome. 3. Menopausal.
4	**Rickets :** Due to :	1. Vitamin D deficiency. 2. Renal Rickets.
5	**Neoplastic**	e.g. Primary tumours of bone or metastasis.

Clinical picture of fracture :

1. History of trauma.
2. Pain localised to the site of the fracture.
3. Swelling due to the fracture hematoma, edema in the surrounding tissue and bony ends.
4. Ecchymosis.
5 .Loss of function of the injured part.
6. Deformity due to displacement of the fragments by :
(a) The causative violence.
(b) The weight of the limb
(c) The action of the muscles.
7. For the complete clinical diagnosis of a fracture, the motor and sensory innervation of the limb, and the peripheral circulation should be examined to record and treat any injury of nerves or blood vessels.

Signs of fracture :

1. Localised tenderness : at the site of the fracture.
2. Deformity.
3. Crepitus : it is a definite sign of fracture. But DO NOT search for it as this may induce more trauma. Absent in cases of incomplete or impacted fracture.

Deformity : (Morbid anatomy) :

1. Shortening - lengthening.
2. Angulation.
3. Over-riding.
4. Rotation especially in spiral fracture.

Radiological diagnosis :

1. Radiographs of the whole bone should be including the joint above and joint below.
2. X-rays should also be obtained in two planes at right angles to each other.

Fractures during childhood have special features :

1.The fracture may be incomplete as in greenstick fracture.
2. The fractures unite in a shorter time than adults.
3. Correction of malunion by growth.
4. Joint stiffness does not occur.
5. The fracture may involve the epiphysial cartilage plate leading to growth retardation.

Treatment of simple fracture

General Management :

1. **ABCDE**
2. **Immobilisation** of the part by a splint e.g. a piece of wood.
3. **Sedation** for the patient e.g. morphine, novalgin.
4. **Tetanus toxoid** and **antibiotics** in compound fracture.
5. **Full examination** to exclude other injuries and treat them, if present.

Local Treatment :

I- Reduction : Aiming at restoration of the continuity of the fracture in the 3 dimensions.

- **Methods :**

[A] Closed reduction : using the following principles :

1. Gravity : e.g. fracture neck or shaft humerus.
2. Closed manipulation : Better under general anesthesia.
3. Traction : e.g. fracture shaft femur.

[B] Open reduction :

- **Indications :**

1. Interposition of soft tissues between the fractured segments.
2. If it is impossible to reduce the fracture by closed method.
3. Intra-articular fractures.
4. Late unreduced fracture.
5. When internal fixation is needed.

II- Fixation or immobilization :

[A] External fixation. **[B] Internal fixation.**

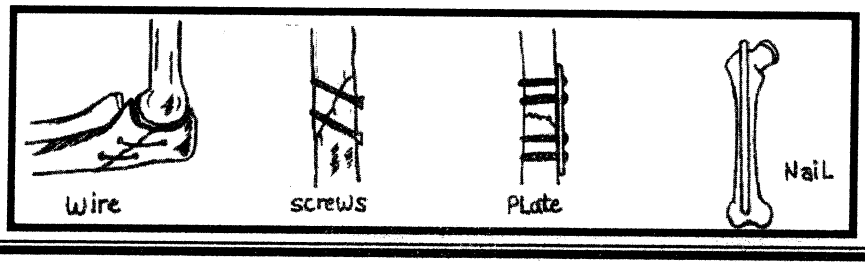
[A] External fixation by :

1. Plaster of Paris. Complications :
(a) Joint stiffness.
(b) Muscular atrophy.
(c) Inability to maintain perfect reduction during the immobilization period.
(d) Liability to cause compartment syndrome or Volkmann's ischemic contracture.
2. Bandage : As in the fracture of the clavicle.
3. Sling : As in fracture of the humerus.
4. Splint.

[B] Internal fixation by :

- Open reduction (by operation) and the bone is fixed in the corrected position by an internal mean.
- This may be by :

1. Suture : by stainless steel, in case of fracture olecranon and patella.
2. Screw : e.g. in oblique fracture of the tibia.
3. Plate + Screw fixation : e.g. in transverse fracture.
4. Nail (intra medullary trifalanged nail).
5. Bone grafts.



Indications of internal fixation :

1. Difficult fractures :

(a) Those prone to non-union e.g. femoral neck.

(b) Those prone to malunion e.g. fracture-sublaxation of the ankle.

(c) Those prone to be pulled apart by muscle action e.g. transverse fractures of patella & olecranon.

2. Pathological fractures.

3. Multiple fractures : With two major fractures in one limb, fixation of one facilitates closed treatment of the other.

4. Nursing difficulties : e.g. Elderly patient.

5. Associated soft tissue injuries : e.g. vessels or nerves.

6. Unstable fractures.

III- Rehabilitation :

- To restore function by active movements which has to start even while the limb is immobilised.
- Active movements are better than passive movements.

Treatment of compound fractures

[A] As regards the fracture :

- Some form of external stabilization by a plaster slab or external skeletal fixators is used.
- Internal fixation should be avoided for fear of inviting infection.

[B] As regards the wound :

- Antibiotics and prophylaxis against tetanus follow the general principles.
- In the theatre under general anaesthesia the wound is thoroughly cleansed.
- Any soft tissue injury is delt with according to the rules.

1. If the wound is clean i.e. no risk of contamination, within 6 hours from the injury we do excision of the wound edges with its closure. Then treat the fracture as a simple one. With proper closed reduction and external fixation.

2. If the wound is contaminated : The wound is left unsutured with a daily dressing till the wound is clean, then 2ry sutures to the edges are done.

Complications of fractures

General complications : (mainly in fracture spine,pelvis & femur)

1. Shock : Neurogenic or hypovolemic shock may accompany major fractures as fracture spine, pelvis or femur. The blood loss in a fracture pelvis may amount up to 2-2.5 litres.

2. Fat embolism

3. Complications of prolonged recumbancy :

(a) Respiratory complications e.g. pulmonary embolism.

(b) Deep vein thrombosis.

(c) Bed sores.

4. Tetanus : In compound fracture.

5. Paralytic ileus & acute gastric dilatation.

Early local complications :

1. Skin injury : The simple fracture becomes compound.

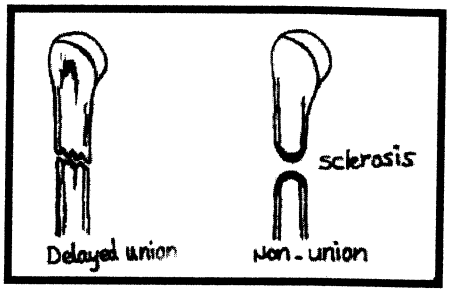
2. Vascular injuries. **3. Nerve injuries.** **4. Tendon or muscle injury.**

5. Infection : This serious complication may complicate compound fractures. It leads to delayed healing, non union or osteomyelitis.

6. Avascular necrosis of bone : Certain fractures may damage the blood supply to some areas of bones leading to avascular necrosis. The commonest example is intracapsular fracture of neck of femur.

7. Visceral complications : Fracture of the pelvis may be complicated by a retroperitoneal hematoma, injury to the bladder or urethra.

Delayed local complications :



[1] Malunion : This means union but with some deformity.

[2] Delayed union and non-union :

Factors affecting union of fractures :

1. Age of the patient :
- Children have rapid healing than adults.
- Elderly people have retarded healing.

2. Type of fracture : Long oblique & spiral fractures heal more rapidly than transverse fractures.

3. Position of the fragments : If the fragments are impacted, union occurs rapidly.

4. Vascularity of the fragments e.g.:
- Intracapsular fracture of neck of femur with injury of blood supply leads to avascular necrosis of the femoral head.
- Fractures of the lower thirds of the tibia and ulna are also liable to delayed union.

5. Immobilization of the fragments.

6. Infection.

Etiology :

General causes : e.g. Old age, debilitating disease, osteomalacia, rickets ... etc.

Local causes :

1. Impaired blood supply of one or both fragments e.g. in intracapsular fracture neck femur.
2. Inadequate immobilization.
3. Interposition of soft tissue between the fragments.
4. Excessive skeletal traction.
5. Infection.
6. Severe initial trauma with compound fracture and extensive damage of the soft tissues:
7. Pathological fractures e.g. due to malignant disease.

Items	Delayed Union	Non Union
X-ray	The bone ends are decalcified. The fracture line is widened into gap full of granulation tissue.	The bone ends are sclerosed. The medullary canal is closed by a dense bone.
Clinical Picture	Clinically there is still abnormal movement and tenderness at the site of the fracture.	
Follow up	There is still a chance of union with prolonged immobilization.	Spontaneous healing is impossible.

Treatment :

1. Internal fixation and application of autogenous bone graft obtained from the cancellous tissue of the iliac crest.
2. Delayed union may be treated by prolongation of immobilization.

[3] Sudek's atrophy :

Definition : This is a syndrome in which there is osteoporosis, swelling of the soft tissue, vascular stasis, pain and joint stiffness complicating a fracture.

Etiology : Unknown, it is supposed to be due to reflex vascular stasis or due to reluctance of the patient to use the limb after removal of the splint.

Incidence : It is most often seen after **Colles' fracture.**

Treatment : Intensive physiotherapy to the patient, analgesics, splinting and sympathetic block.

[4] Myossitis ossificans :

Definition : Hetrotropic bone formation in the fleshy part of the muscle.

Incidence : Most often seen after dislocation of the elbow, shoulder, hip or supracondylar fracture.

Pathogenesis : The hematoma in the injured muscles communicating with the fracture hematoma either undergo metaplasia in the fibroblasts which change to osteoblasts or osteoblasts from the periostium migrate from the bone to the muscle hematoma. Calcium will be deposited later and become calcified then it becomes ossified.

Treatment :

1. Prophylactic treatment : Proper treatment of the supracondylar fracture preventing the development of haematoma and proper fixation of the fracture.
2. If the ossification develops : prolong the period of immobilisation. This gives the chance for the body to remove it almost completely. **No massage is allowed** as this may increase the ossification.
3. Residual mass after prolonged immobilisation can be removed surgically.

[5] Volkmann's ischemic contracture :

(**Contracture** = permanent shortening of the muscle due to its replacement by fibrous tissue).

Etiology :

- Injury to the arterial supply of the muscles of the limb leads to ischemia & gradual death of these muscles & its replacement by fibrous tissue. This is common around the elbow & the knee.

Clinical picture :

Early : Manifestations of acute ischemia in the form of **P**ain, **P**allor, **P**aralysis, **P**arasthesia, **P**ulselessness.

Later : The patient will present with the typical deformity of : flexion at the wrist joint, extension at the metacarpophalyngeal joins, flexion at all the inter-phalangeal joints. Extension of the wrist leads to more flexion at the interphalangeal joints. With flexion at the wrist, the surgeon can passively extend the finger.

Treatment of Volkmann's Contracture :

1. Prophylactic treatment : If the patient develops acute ischemia with the **5(Ps)** of **P**ain, **P**allor, **P**aralysis, **P**arasthesia, **P**ulselessness. The plaster is removed and the fracture is recorrected, and the patient is given vasodilators. If no immediate response is obtained, the vessel is exposed and dealt with according to the type of injury present.

2. In established case :

(a) Early : Do passive traction of the shortened muscles in the hyper extended position with splintage in this position.
(b) Late : Sliding the origin of the flexor muscles from the medial epicondyl downwards to the ulna, extra length is given to the shortened muscles correcting the shape but not the function of this hand.

[6] Joint stiffness : Certain joints as the elbow, shoulder and hip are particularly prone to this problem. Open reduction and internal fixation avoids this complication.

[7] Growth disturbance : May occur if the fracture affects the epiphysial plate in children.

[8] Osteoporosis : Due to prolonged immobilization.

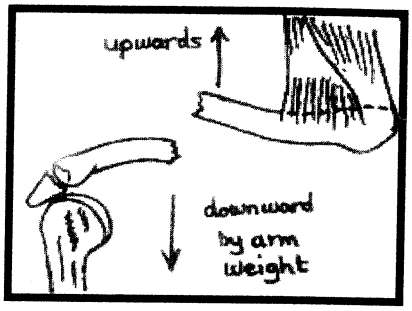
Fractures of the upper limb
Fractures of the clavicle

[A] Fracture of the middle third (shaft) : 80%

This is the commonest fracture in the whole body.

- **Why the middle third ?**

1. It is the thinnest part of the bone.
2. The junction of the two main curves of the shaft.
3. Site of insertion of subclavius.
4. Site of entrance of nutrient artery.



Classification :

- In children the fracture is often of the greenstick variety.

Mechanism of injury :

1. It is due to fall on out-stretched hand.
2. Less commonly due to direct blow or to a fall on the point of the shoulder.
3. Rarely pathological.

Morbid anatomy : The weight of the arm leads to downwards displacement of the outer fragment & the sternocleidomastoid muscle leads to upwards displacement of the proximal fragment.

Clinical picture : (As General)
The shoulder is dropped and deformed, and the patient usually supports the elbow with the opposite hand and bends his head to the affected side to relax the sternomastoid muscle.

Complications : (As General)

1. Skin.
2. Muscle : Tear of subclavius muscle.
3. Vessel : Injury to subclavian artery or compression of subclavian vein.
4. Brachial plexus injury.
5. Injury to the dome of the pleura and apex of the lung leading to pneumothorax.
6. Injury to the thoracic duct (on the left side).
7. Mal-union is very common (of no functional significance).
8. Non-union is rare due to its rich blood supply.

Treatment :

- No attempt should be made to reduce the fracture, because it is not possible to hold the reduction even if it is achieved. Most surgeons rely only on a broad arm sling (for 3 weeks) and analgesics.
- Internal fixation may be needed when associated with vascular or nerve injury.

[B] Fracture of the outer third : 15%

Usually no displacement occurs because both fragments are attached to the scapula by ligaments. If there is no displacement, no treatment is required (but analgesics).

[C] Fracture of the inner third is rare : 5%

Fractures of the scapula

[1] Fracture of the neck of scapula :

Here, the fracture results from direct violence. The fracture line usually runs from the suprascapular notch to below the coracoid process.

Treatment : By application of a broad arm sling and early active shoulder movement as pain subsides.

[2] Fracture of the body of scapula :

This fracture is also the result of direct violence. The fracture line may be stellate. Comminution is common.

Treatment : The same as above. It is important to look for and exclude an associated chest injury.

Dislocation of the shoulder joint

Dislocation of the shoulder joint may be :

1. Traumatic.
2. Recurrent.
3. Pathological.

Instability of shoulder joint is due to :

1. The shallowness of the glenoid cavity.
2. The laxity of the capsule.
3. The lack of support by strong ligaments and muscles.
4. Big head of the humerus.
5. Wide range of movement.

[A] Anterior dislocation :

Morbid anatomy :

The head of the humerus usually dislocates forwards to assume one of the following positions :

1. Subcoracoid (commonest).
2. Subglenoid.
3. Luxatio recta is an anterior dislocation in which the arm is abducted above the head.
4. Subclavicular.

Trauma :

- The injury is produced by forced extension and external rotation of the abducted arm.
- The head of the humerus is forced against the capsule of the shoulder and either ruptures its anterior aspect or avulses the labrum glenoidal from the glenoid.

Clinical picture : (As General)

1. Deformity :

- The shoulder is flattened.
- The arm appears to take origin from a point under the junction of the middle and outer thirds of the clavicle.

2. Swelling : The head is felt displaced in the subcoracoid region.

3. Loss of function : The shoulder cannot be moved and the arm is held in a position of slight abduction.

X-rays : In anteroposterior views are diagnostic.

Treatment :

Under general anaesthesia with muscle relaxation closed reduction of the dislocated head is done using the **Kocher's technique** the principles of which are :

1. Pulling downwards to get the head in the subglenoid position.
2. Externally rotate the arm to put the subscapularis muscle at the stretch to get it fatigued.
3. Then the limb is adducted till it comes in contact with the front of the chest.
4. Then the operator does internal rotation till the hand of the patient touches the healthy shoulder.

- Reduction is confirmed by putting the shoulder through a full range of movement under anesthesia.
- The arm is then immobilized by sling & bandaged in adduction & internal rotation of the shoulder for 3 weeks to allow healing of the torn capsule& to minimize the tendency for recurrence.
- At the end of 3 weeks, the sling is removed and progressive mobilization of the shoulder is done avoiding only external rotation in abduction. (The position in which the shoulder dislocates).

N.B. The older Hippocrates closed technique is rarely used except in cases of neglected dislocations. It entails traction on the arm with counter-pressure by the unshoed foot of the operator in the axilla.

Complications :

1. Axillary nerve injury.
2. Avulsion of the supraspinatus tendon. The patient is unable to initiate abduction.
3. Fracture of the greater tuberosity or the neck of the humerus.
4. Recurrence (common problem). Due to detachment of the labrum glenoidal and the anterior capsule from the anterior margin of the glenoid. A pouch is formed into which the humeral head slips easily.

- **Clinically :** Redislocation occurs with increasing ease and frequency following relatively minor everyday actions such as combing the hair.
- **Reduction** is equally easy and usually the patient can reduce the dislocation himself.
- **Treatment :**

1. Putti-Platt operation : The idea is to limit external rotation of the shoulder by capsulorraphy and shortening of the subscapularis muscle by overlapping.
2. Bankart operation : Has a better result. The original lesion in the capsule or labrum glenoidal is repaired.

[B] Posterior dislocation :

- This is not common. The head slides backwards to lie below the acromion (subacromial dislocation) or in the infraspinous fossa of the scapula (subspinous dislocation). It is usually caused by forced internal rotation of the abducted arm.
- It should always be suspected after an epileptic fit or an electric shock.

Fractures of the humerus

- The fracture may involve :

[A] The proximal humerus :

1. Surgical neck.
2. Anatomical neck.
3. Greater tuberosity.

[B] The shaft.

[C] The distal end of the humerus.

Fractures of the Proximal Humerus

Type of the patient : Common in the elderly.

Classification :

1. Greater tuberosity.
2. Lesser tuberosity.
3. Surgical neck.

The classification then identifies the fractures as :

One-part fracture (80%)	No displacement (even though a fracture line may exist between any number of segments).
Two-part fracture	One segment is displaced.
Three-part fracture	Two segments are displaced.
Four-part fracture	Four segments are displaced.

Mechanism of injury :

1. Indirect trauma : Fall on outstretched hand.
2. Direct trauma (Less common).

Clinical picture : (As General)
The patient complains of pain in the shoulder and inability to move the joint. In minor impacted fractures of the surgical neck, limited movement may be possible.

Treatment :

[1] One-part fractures : External immobilization using a sling with early active mobilization of the shoulder to avoid stiffness.

[2] Two-part fractures :

(a) Fractures of greater tuberosity : open reduction & internal fixation (using screw or wire) is mandatory.
(b) Fractures of lesser tuberosity : usually requires just immobilization.
(c) Fractures of surgical neck : Application of a collar & cuff to allow gravity to reduce the fragments.

[3] Three-part fractures : Open reduction and internal fixation.

[4] Four-part fractures : These have a high incidence of avascular necrosis of the humeral head and are probably best treated by prosthetic replacement of the head.

Complications :

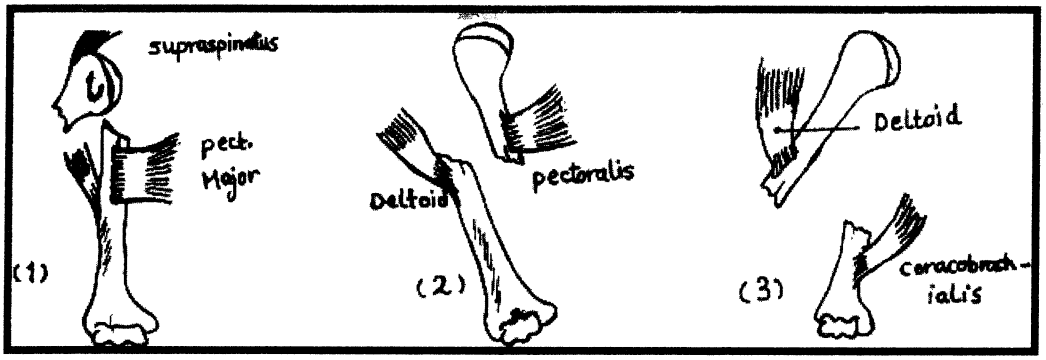
1. **Shoulder stiffness :** common especially in the elderly
2. **Axillary nerve injury.**
3. **Malunion** is not uncommon but compatible with excellent functions in many cases.
4. **Avascular necrosis** of the humeral head.
5. **Non-union** (not common).
6. **Dislocation of the shoulder.** In these cases the dislocation should be treated first.

Fracture of the Shaft of the Humerus

Mechanism of injury :

1. Indirect trauma : Fall on outstretched hand.

2. Direct trauma : Direct blow over the shaft producing a transverse fracture.



Morbid anatomy : This depends on the level of the fracture :

1. Just below the surgical neck : The proximal fragment is abducted by the supraspinatus and the distal fragment is adducted by the pectoralis major.

2. Middle third : The upper fragment is adducted and pulled inwards by the pectoralis major and the lower is abducted by the deltoid.

3. Below the deltoid tuberosity : The proximal fragment is abducted by the deltoid and the distal fragment is adducted and pulled upwards by the coracobrachialis.

Clinical picture : (As General)

Treatment :

[I] Closed Method :

1. Reduction :

(a) In fracture above insertion of the deltoid : the distal part is adducted.

(b) In fracture below insertion of the deltoid : the distal part is abducted.

2. Fixation : U-shaped plaster is applied to the arm and the limb is supported by a collar and cuff sling. The weight of the limb will maintain the 2 fragments opposite each other while the plaster cast will prevent side movements.

3. Rehabilitation : Early movement of the wrist and finger joints.

[II] Open Method :

Occasionally, it is not possible to obtain a satisfactory reduction by the closed methods. One should then proceed to open reduction and internal fixation using a plate, or if the fracture is transverse and near the middle of the shaft, an intramedullary nail.

Complications : (As General)

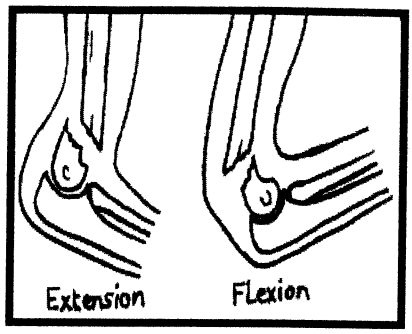
1. Radial nerve injury.
2. Delayed union.
3. Non union due to injury of profanda artery & nutrient arteries.
4. Joint stiffness.

Fractures of the distal end of humerus

1. **Supracondylar.**
2. **Intercondylar (T or Y).**
3. **Condyle :** Lateral and medial.
4. **Articular surface :** Capitulum and trochlea.
5. **Epicondyl :** lateral and medial.

Supracondylar fractures

[A] Supracondylar fractures in children :



Mechanism of trauma :

1. Extension Type (99%) : As a result of fall on outstretched hand with the elbow slightly flexed : the distal fragment is displaced posteriorly.
2. Flexion type (1%) : As a result of a fall on a flexed elbow.

Classification :

- The fracture line runs transversely through the distal metaphysis of the humerus.
- The fracture is greenstick in 50% of cases.

Morbid anatomy :

In the extension type, displacement usually consists of the following elements :
1. **Backward shift** of the distal fragment.
2. **Pronation** of the distal fragment because the hand is usually pronated at time of the trauma.
3. **Internal rotation** of the distal fragment as pronation produces internal rotation.
4. **Adduction** of the distal fragment. Due to pull of the arm muscles.

Clinical features :

1. There is pain and swelling of the elbow region (above the line of joint). Swelling occurs rapidly so that it may obscure the other physical signs within 3 to 4 hours.
2. The child is not able to move the elbow. (There may be abnormal movement).

Examine the hand for acute ischemia.

Complications :

1. **Nerve injury.** Damage to the median, ulnar and radial nerves may occur. Conservative treatment is satisfactory in most of the cases.
2. **Vascular injury.** The brachial artery may be injured at the time of the trauma or more commonly compressed by swelling and flexion following reduction.
3. **Elbow stiffness.**
4. **Malunion** may result from failure to reduce the fracture properly. It consists of residual cubitus varus and internal rotation.
5. **Myossitis ossificans.**
6. **Volkmann's ischemic contracture.**

Treatment :

[I] Undisplaced fractures and Greenstick fractures : No manipulation. Fixation using a posterior plaster slab and collar and cuff with the elbow flexed for 3 weeks.

[II] Displaced fractures :

[1] Closed method of treatment.

[2] Open method of treatment :

Open reduction and internal fixation (using wires) are indicated when the closed method fails to obtain satisfactory reduction.

Principles of the technique :

[A] Reduction :

1. Traction and counter traction in the long axis of the forearm.

> Extension of the elbow should not be attempted until traction has drawn the distal fragment beyond the proximal fragment. This precaution reduces the chance of injuring the brachial artery by the distal end of the proximal fragment when the elbow is subsequently extended.

2. Extension of the elbow is next done while maintaining traction.
3. Supination of the forearm, to correct the pronation deformity.
4. Carrying angle is restored.
5. Elbow is slowly flexed until a point just beyond 90° is reached.

Throughout this manoeuvre, the radial pulse is felt and if it is obliterated by flexion the elbow is extended until the pulse returns.

[B] Fixation :

Above elbow posterior slab is applied. A collar and cuff is added.

[C] After care :

- The child should be admitted to the hospital for observation of the circulation to the hand (usually for 48 hours).
- If at any time the circulation is felt to be impaired, the slab is removed and the elbow is extended.
- If there is no improvement, the brachial artery should be explored.
- The fracture unites in 3 or 4 weeks.

Some surgeons prefer to fix these fractures with the elbow in extension as :
1. It allows better control of the carrying angle.
2. Reduces the risk of impeding the circulation to the forearm and hand.

[B] Supracondylar fractures in adults :

- These are much less common than in children. However, the principles are almost the same.

Complications of injury around the elbow joint

1. Tear in the brachialis muscle.
2. Traumatic myositis ossificans.
3. Volkmann's ischemic contracture.
4. Brachial artery injury.

5. Nerve injury : ulnar, median or radial.

- **Ulnar nerve injury :** Is either immediate or delayed due to its irritation by the rough callus or its entanglement in fibrous tissue or due to cubitus valgus (malunion of a supra condylar fracture associated with increase in carrying angle where the ulnar nerve is stretched).

6. Bony complications :

- Delayed or non union.
- Malunion

7. Articular complications :

- Hemoarthrosis.
- Ankylosis (Stiffness).
- Later osteoarthritis.

Dislocation of the elbow

Trauma & morbid anatomy :

- This usually occurs in a posterior direction, following a fall on the outstretched hand with the elbow slightly flexed.
- The coronoid process of the ulna passes posteriorly below the distal end of the humerus.
- Spasm of the triceps muscle then locks the elbow in a position of posterior dislocation.
- The dislocation can occur in other directions, anterior, medial or lateral. On occasions, the radius or ulna dislocate in opposite directions (divergent dislocation).

Clinical picture :

1. Loss of function : The patient is unable to move the elbow from a position of slight flexion.
2. Swelling : The olecranon can be felt to be abnormally posterior to the humeral condyles.
3. Measurements : The triangle formed by the tip of the olecranon and the condyles is no longer equilateral (DD supracondylar fracture humerus).

Treatment :

- Under general anaesthesia traction is applied in the long axis of the slightly flexed ulna.
- The joint should be immobilized for 3 weeks in an above elbow posterior slab to allow for healing of the capsule and the ligaments.
- Irreducible dislocations are rare and are treated by open reduction.

Complications :

1. Associated fracture of the radial head or coronoid process.
2. Median or ulnar nerve damage.
3. Vascular damage is unusual.
4. Myositis ossificans.

Fractures of the forearm

These are classified as follows :

1. Fractures of the proximal ulna : Olecranon and coronoid.
2. Fractures of the radial head.
3. Fractures of the shafts of radius and ulna.
4. Monteggia fracture.
5. Galeazzi fracture.

Fractures of Proximal Radius

2. Fractures of the head of the radius

Trauma and morbid anatomy :

A fall on the outstretched hand may result in one of the following fractures to the head of radius :

1. Fissure fracture.
2. Marginal fracture without or with displacement.
3. Comminution of the head.

Clinical picture :

The clinical signs are often minimal and do not suggest that a fracture has occurred. There is commonly very little bruising and minimal swelling. The only significant physical signs are painful limitation of movement combined with tenderness over the head of the radius. The injury is revealed radiologically.

Treatment :

1. Undisplaced fractures : are treated by a sling till pain decreases and then active movement should be encouraged.
2. Displaced fractures : may be either treated by excision of the radial head, internal fixation or by conservative measures as the undisplaced variety.
3. Comminuted fractures : should be treated by early excision of the whole head to prevent adhesions and joint stiffness.

3. Fractures of the Shafts of Radius and Ulna

[A] Fracture of one bone without angulation :

This is caused by direct trauma. It requires immobilization in an above elbow plaster for 6 weeks in an adult and for 3 weeks in a child.

[B] Fracture of both bones :

1. Direct blow will break the bones at approximately the same level.
2. Indirect trauma results in oblique fractures at different levels.

Morbid anatomy :

- If the fracture occurs above the insertion of the pronator teres, the proximal fragment is pulled into supination by the biceps and supinator muscles.
- If the fracture is below the insertion of the pronator teres, the proximal fragment is in mid-pronation.

Treatment :

1. Undisplaced fractures : are treated by a full arm plaster cast with 90□ elbow flexion and neutral rotation, for approximately 6 - 9 weeks.
2. Displaced fractures :

- **In adults :** open reduction & Internal fixation (above elbow plaster cast may be added for 6 weeks).
- **In children :** closed reduction and plaster fixation is usually successful. (As residual angulation of about 5 degrees can be accepted). If not, treat as adults.

Complications :

1. Non-union (rare).
2. Malunion.
3. Nerve injury is uncommon.
4. Acute compartment syndrome (treated by fasciotomy).
5. Synostosis (cross - union) between radius and ulna.

4. Monteggia Fracture-Dislocation

- This consists of fracture of the ulna and dislocation of the superior radio-ulnar joint.

Treatment :

Open reduction and internal fixation of the ulna and closed reduction of the radial head. This is followed by the application of a full-arm plaster cast for 6 weeks.

5. Galeazzi Fracture-Dislocation

It consists of a fracture of the radius and dislocation of the inferior radio-ulnar joint.

Fractures of distal ends of radius & ulna

These are classified as follows :

Adults :

1. Colles' fracture.
2. Smith's fracture : The reverse of Colles'.
3. Barto's fracture : Intra articular fracture of the distal radius.
4. Fracture of the radial styloid process.

Children :

1. Fracture of the distal radial epiphysis.
2. Fracture of the distal radial metaphysis.

Colles' fracture

Definition :

- This is a fracture of the distal end of the radius produced by a fall on an outstretched hand.

Type of patient :

- It is not common below the age of 50.
- After this age it becomes increasingly common particularly in women de to postmenopausal osteoporosis.

Mechanism of injury :

- Fall on the palm of outstretched hand.

Morbid anatomy :

- The fracture line is within one inch of the lower end of the radius in its cancellous part. It runs obliquely upwards and backwards.

- **The distal fragment shows the following displacement :**

1. Dorsal displacement and angulation.
2. Radial displacement and angulation.
3. Upward displacement and impaction.
4. Supination.

- There is always an associated injury to the inferior radioulnar joint.

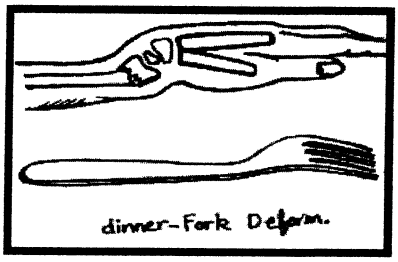
Clinical features and diagnosis :

- The displacement causes the characteristic **"diner-fork"** deformity.

The lower fragment will be impacted and displaced backward and laterally.

- The radial & ulnar styloid processes are approximately at the same level, (normally the radial is distal).

- X-ray is diagnostic.



Treatment :

[A] Reduction :

Under anesthesia. The manipulation is carried out by 3 grips :

1. Traction along the long axis of the limb in order to disimpact the fragments.
2. Pressing the distal segment anteriorly to correct the posterior displacement.
3. Pushing the distal fragment medially to correct the radial displacement.

[B] Fixation :

A below elbow plaster cast is applied holding the wrist in palmar flexion and ulnar deviation.

[C] After care & rehabilitation :

- The circulation to the fingers should be watched in the initial 24 hours.
- The fingers should be actively mobilized immediately after the injury to prevent stiffness.
- This is in fact more important than the treatment of the fracture itself regarding functional outcome.
- The plaster is maintained for 6 weeks.

Complications :

1. Skin injury : leading to compound fracture.

2. Tendon injury :

(a) Rupture.

(b) Fraying of the extensor pollices longus tendon and later rupture.

3. Vascular injury : radial or ulnar artery injury.

4. Malunion : While union is the rule, there is often some residual deformity because :

(a) Redisplacement may occur inside the cast.

(b) A Colles' plaster does not control the supination element of the deformity.

> In most cases exercises and physiotherapy is all that is needed.

5. Madelung deformity : It occurs in young patients where there is arrest of growth of the radius. The wrist and the hand will deviate to the radial side.

6. Nerve injury e.g. :

(a) Median nerve paralysis.

(b) Carpal tunnel syndrome : This is a late complication which is treated by surgical division of the flexor retinaculum after confirmation of the diagnosis by nerve conduction studies.

7. Stiffness of the wrist, shoulder and fingers.

8. Sudek's atrophy.

N.B. Smith's Fracture :

This is the reverse of Colles' fracture. It is due to fall on the dorsum of the wrist.

Injuries of the hand

Injuries of the carpus

Any of the bones of the carpus may be fractured or dislocated. Fracture of the scaphoid bone is the commonest injury of the carpus (60-70%).

Fractures of the Scaphoid

The injury occurs following a fall onto the outstretched hand typically in young adults.

The fracture may occur at :

1. The waist of the bone (commonest site) as the bone is bent at its wrist over the radial styloid process.
2. The proximal third (proximal pole fracture).
3. The distal tubercle.

Clinical picture and radiology :

- There is **pain in the wrist,** but function of the wrist may not be grossly impaired. On examination there is **tenderness** over the scaphoid in the anatomical snuff box, a little **swelling** and **no bruising.**
- These physical signs often appear trivial and suggest a sprained wrist rather than a fracture.
- Radiological examination including (AP, lateral and oblique views), may not be informative immediately after the injury because no displacement occurs at the fracture site.

Treatment :

- Because this fracture does not produce obvious clinical and radiological signs, it is often missed and wrongly diagnosed as a **"sprained wrist".** The wrist is then treated with strapping and as a result the fracture may not unite.
- Therefore, whenever suspected a scaphoid plaster should be applied for 2 weeks. Two weeks after the injury, the plaster should be removed and the wrist re-X-rayed. By this time bone resorption should have occurred at the fracture site which should be radiologically evident.
- The wrist is fixed in a below elbow plaster cast including the proximal phalanx of the thumb for 8 weeks. This will result in bony union in 90% of uncomplicated cases.

Complications :

1. Non-union is treated by bone grafting with or without internal fixation.
2. Avascular necrosis of the proximal fragment may occur because the blood supply to the proximal part of the bone often enters entirely through the distal pole.

Fractures of the Base of the First Metacarpal

They are especially common in boxers from blows or falls on the radial side of the hand.

The fracture may be :

1. Transverse with no joint injury and no tendency to displacement **(simple fracture).**

2. Oblique (in the thumb) with involvement of the articular surface and constant tendency to redisplacement from retraction of the flexor and extensor tendons of the thumb **(Bennet's fracture - dislocation).**

Treatment :

Simple fractures are reduced closely and immobilized in plaster. Bennet's fracture of the first metacarpal may need internal fixation.

Fractures of the Shafts of the Metacarpals

They result from blows or falls upon the hand. They are treated by a posterior plaster slab for 3 weeks.

Fractures of the Necks of the Metacarpals

They are due to blows on the closed fist. The head of the bone is usually tilted forwards towards the palm. The deformity is corrected by backwards pressure along the flexed finger and a dorsal plaster slab holding the finger in flexion is applied for 3 weeks.

Fracture of the phalanges

These are usually transverse and the fragments are often angulated forwards by the tension of the lumbrical and interosseous muscles. The angulation is corrected and the finger is immobilized in semiflexion over a finger wire splint incorporated in a forearm plaster cast.

Mallet Finger

This is disinsertion of the extensor expansion from the base of the terminal phalanx resulting in flexion deformity of that phalanx. The patient cannot actively extend the distal interphalangeal joint.
The finger is immobilized with the proximal interphalangeal joint in flexion (so as to relax the lateral slip of the extensor expansion) and the distal interphalangeal joint hyperextended (so as to approximate the end of the tendon to the raw area). This is maintained for 6 weeks.

Injuries of the pelvis & lower limbs
Fracture of the pelvis

Mechanism of injury :

1. Direct anteroposterior or lateral compression from a crushing injury, e.g. in road traffic accident.
2. Indirect lateral compression through the lower limb.
3. Rarely, falls in the home in elderly patients.

Classification :

[A] Fractures outside the pelvic ring :

1. Traction injury e.g.:
   - AIIS due to avulsion by the rectus femoris tendon.
   - Ischial tuberosity due to avulsion by the hamstrings.
   - ASIS due to avulsion by the sartorius.
2. Iliac wing.
3. Sacrum.
4. Coccyx. Usually bed rest is enough.

[B] Single fracture of the pelvic ring.

[C] Double fractures of the pelvic ring :

1. Combined breaks in both the anterior and posterior segments.
2. Double breaks in the anterior segment.

[D] Lateral wall fracture :

1. Acetabular fracture.
2. Segmental fracture.

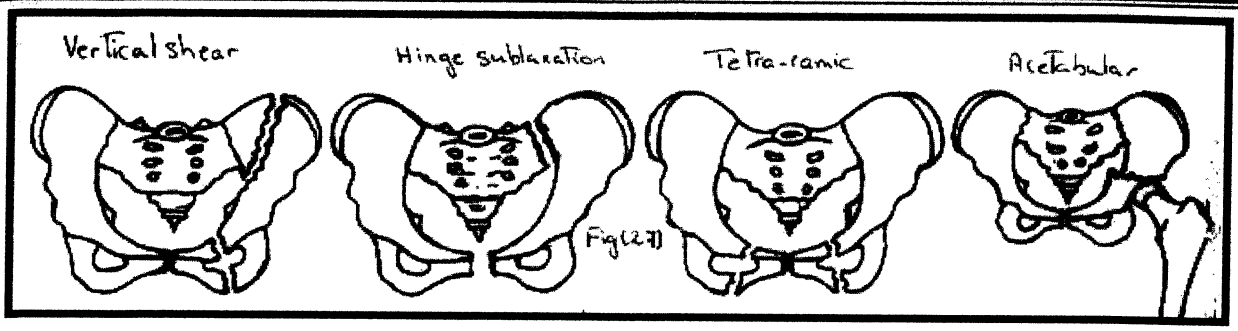
Double fracture of the pelvic ring

1. Combined breaks in both anterior and posterior segments of the pelvic ring :

Items	Vertical shear fracture	Hinge sublaxation
Consist of	Fractures of both rami or disruption of the symphysis pubis at the front and fracture of the ileum or sacrum or a disruption of the sacroiliac joint posteriorly.	Disruption of the symphysis pubis with wide separation in addition to disruption of one sacroiliac joint with little separation (open-book injury).
Displacement	The mobile hemipelvis tends to be pulled upwards by the anterior and posterior trunk muscles. It also opens up like a book by the weight of the leg.	The stability of this injury is variable.
Prognosis	This type of pelvic fracture is associated with the highest mortality and morbidity.	Variable.

2. Double breaks in the anterior segment of the pelvic ring :

Fractures of the pubic rami on both sides **(Tetraramic fractures),** the detached segment may be displaced backwards. The urethra is often sheared off at the apex of the prostate in a male.



Lateral wall fractures

1. Acetabular fractures :

(a) Linear fractures.
(b) Comminuted fracture.

- **They result in sublaxation or central dislocation of the femoral head.**

2. Segmental fracture :

- **This occurs when segment of the lateral wall of the pelvis containing the intact acetabulum is displaced inwards.**

Clinical picture :

[A] Generally :

1. The patient is usually shocked due to blood loss inside the pelvis.
2. Retroperitoneal hemorrhage may cause ileus and simulates injury to viscera.

[B] Locally :

1. There is pain, in and around the pelvis.
2. The patient cannot stand lifting his legs but passive movements at the hips can be elicited.
3. The leg may be externally rotated.
4. A gap may be felt at the symphysis pubis.

Complications of pelvic fracture :

[A] Generally :

1. **Shock.** Blood loss from a fracture pelvis may amount to 2.5 liters.
2. **Paralytic ileus** due to retroperitoneal haematoma.
3. **Deep vein thrombosis** is mainly due to prolonged bed rest
4. **Complications of prolonged recumbancy.**

[B] Locally :

[A] Injury of the viscera :

1. The male urethra.
2. The urinary bladder. If there is any doubt about a vesical or urethral injury, an ascending urethrocystogram is performed.
3. The rectum detected by per rectal examination.
4. The vagina.
5. Nerve roots, so nervous examination should be performed.
6. Pelvic arteries and veins.

[B] Malunion causes no significant problems except in females (cesarean section may be required for delivery).

[C] Secondary osteoarthrosis commonly occurs after disruptions of the sacroiliac joints & acetabular fractures.

Management : As general

- **Treatment of visceral injury should take priority over treatment of bony injury.**

<table>
<tr><th>Double fracture</th><th colspan="2">Lateral wall fracture</th></tr>
<tr><td rowspan="2"><u>1. Vertical shear :</u>
• Skeletal traction to the leg.
• Application of a pelvic sling to reduce outward rotation of the hemi-pelvis.
• Traction is maintained for 6 weeks followed by non-weight bearing on the injured side for 6 weeks.</td><th>Acetabular</th><th>Segmental</th></tr>
<tr><td rowspan="2"><u>1. Linear fractures :</u>
<u>A. Undisplaced fracture requires :</u>
Traction for 6 weeks followed by non-weight bearing on the injured side for 6 weeks.
<u>B. Displaced fracture requires :</u>
• Open reduction and internal fixation. <u>OR :</u>
• Combination of longitudinal and lateral skeletal traction.</td><td rowspan="2">Traction for 12 weeks.</td></tr>
<tr><td><u>2. Hinge sublaxation :</u>
• Closed reduction by a pelvic sling or.
• Open reduction and fixation using a plate applied to the superior aspect of the symphysis.
• No weight bearing for 12 weeks.</td></tr>
<tr><td><u>3. Tetraramic fracture :</u>
Uncomplicated fractures require bed rest for 6 weeks.</td><td><u>2. Comminuted fracture :</u>
Traction for 8-12 weeks.</td><td></td></tr>
</table>

Dislocation of the hip

Causes of stability of the hip joint :

1. The depth of the acetabular cavity.
2. The strong support afforded by its ligaments and muscles.
   - Therefore, this injury is rare.
   - The dislocation may be posterior (by far the commonest), anterior or central.

I- Posterior dislocation

Trauma :

This occurs when the hip is flexed and adducted because in this position the head of the femur is covered posteriorly by the capsule rather than bone.

There are two common accidents :

1. A weight falling on the back of a person in a stooping position, e.g. **"fall of roof".**
2. A front seat passenger In car accidents

Morbid anatomy :

The head is dislocated backwards over the posterior lip of the acetabulum. The head escapes into the sciatic notch (sciatic dislocation), & then may pass up onto the dorsum of the ilium (dorsal dislocation).

Clinical picture :

1. Severe pain.
2. Deformity : The hip is in the position of flexion, adduction and internal rotation deformity.
3. Swelling : The femoral head is palpable in the gluteal region.
4. Loss of function : Movements of the hip are painful & limited. There is shortening of the affected limb.

Radiology :

- The femoral head lies outside the acetabulum.
- **Shenton's line is interrupted** (this is the curve formed by the lower margin of the superior pubic ramus and the lower border of the femoral neck).
- Associated fractures may be detected.

Treatment :

- Closed reduction is done under general anaesthesia:
- The hip and the knee are flexed, bringing the head of the bone posterior to the acetabulum.
- The femur is then adducted and pulled vertically upwards so as to draw the head forwards into its socket.
- The hip is then extended and the reduction is maintained by skin traction for 3 to 6 weeks.

Complications :

1. Sciatic nerve injury. The whole nerve may be affected or, the lateral popliteal component.
2. Avascular necrosis of the femoral head. The important factor is to reduce the dislocation as early as possible after the injury.
3. Associated fractures. These should be treated after reduction of the dislocation.
4. Secondary osteoarthritis.

II- Anterior dislocation

Trauma :

Fall of a heavy weight on the upper lateral side of the thigh while the hip is abducted, extended and externally rotated.

Morbid anatomy :

The dislocated head may be forced through :

1. The lower part of the capsule to lie on the obturator externus muscle (obturator dislocation).
2. The anterior part of the capsule to lie on the horizontal ramus of the pubis (pubic dislocation).

Clinical picture :

- **Deformity :** the hip is extended, abducted and externally rotated.
- **Swelling :** femoral head is palpable in the groin.
- **Measurements :** The limb is longer than the normal side.

Treatment :

Closed reduction is usually successful.

III- Central dislocation

It is usually the result of a blow on the greater trochanter pushing the head of the femur through the floor of the acetabulum. It results in a comminuted fracture of the acetabulum and may be accompanied by other fractures of the pelvis.

Treatment :

- Closed and open methods of reduction are both difficult and unsatisfactory.
- The best form of management is to accept the displacement and mobilize the hip with tibial skeletal traction as soon as pain allows.

Fractures of the femur
Fracture of the neck of femur

I- Intracapsular fractures of femoral neck

These occur in that portion of the neck of the femur which lies within the capsule of the hip.

Trauma :

The fracture occurs most commonly in the elderly, especially women, because of senile osteoporosis. The trauma may be a trivial twisting injury.

- There is evidence that the fracture is due to bone fatigue i.e. that the patient falls because the bone fractures, not that the bone fractures because the patient falls.

Morbid anatomy :

They are anatomically divided into :

1. **Subcapital :** occurring at the junction of the head and neck.
2. **Transcervical :** occurring somewhere in the neck.

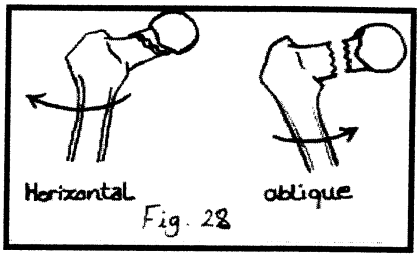


Fig. 28

The fracture line may be :

1. **Horizontal :** the distal fragment will be abducted and impacted.
2. **Oblique to vertical :** The distal fragment will be adducted and displaced.

The fragments are subjected to the opposing forces of the body weight (trying to push the head down) and the pull of the gluteal muscles (trying to pull the neck upwards). Therefore, the fracture is only stable after reduction and internal fixation.

Clinical features :

[A] In unimpacted fractures :

1. The hip is adducted and externally rotated (by the weight of the limb).
2. The greater trochanter is raised.
3. The patient is unable to raise the leg.
4. Movements of the hip are painful.
5. Shortening of the limb is present.

[B] In impacted fractures :

1. No rotation occurs.
2. The fracture appears to be abducted
3. There may be no abnormal physical signs.
4. These fractures are missed clinically and even on X-ray examination.

Treatment :

[A] Treatment of impacted fractures (in all ages) :

- No reduction is required.
- Internal fixation, preferably with plate and screw.

[B] Treatment of displaced fractures :

1. Patient under 65 years : Closed reduction followed by internal fixation. If closed reduction cannot be obtained, open reduction should be performed.

2. Patient over 65 years : The treatment of choice is replacement of the head and neck of the femur with prosthesis (hemiarthroplasty). As avascular necrosis is common and the patient cannot withstand a second operation.

- **In all cases, postoperative early mobilization is required.**

Complications of intracapsular fracture of the neck of femur : As general

[A] Avascular necrosis : (15-35%)

- **Blood supply of the head of the femur :**

1. An extracapsular arterial ring located at the base of the neck.
2. Ascending cervical branches of the extracapsular ring lie subsynovial on the surface of the neck. (Retinacular vessels).
3. Arteries of the ligamentum teres which are not always patent.
4. Terminal branches of the ascending nutrient arteries present within the neck.

- **Sequelae :**

1. Delayed or even non-union.
2. Secondary osteoarthritis of the hip.

[B] Non-union may occur due to :

1. Adequate immobilization is difficult.
2. Poor blood supply to the proximal fragment.
3. The presence of little or no periosteum or surrounding soft tissues.
4. The synovial fluid inside the capsule may lyse the blood clot.

- **Treatment :**

1. In the elderly : Total hip replacement.
2. In young patients : If the head is viable with an intact blood supply (as proven by a bone scan), internal fixation should be repeated and a bone graft inserted.

[C] Thromboembolism : in 25% of patients.

[D] Mortality : may occur in elderly patients.

II- Extracapsular fracture of proximal femur

They are divided into :

1. Trochanteric fractures down to the level of lesser trochanter.
2. Subtrochanteric fracture from the lesser trochanter to 8 cm below.

Trauma :

- **In young persons :** major trauma.
- **In the elderly :** trochanteric fractures may occur secondary to a fall on the side producing a blow over the greater trochanter.

Morbid anatomy : Extracapsular fractures differ from intracapsular ones in 2 aspects :

1. The blood supply of the two fragments is good & so avascular necrosis & non union don't occur.
2. The proximal fragment can be controlled conservatively & so operative treatment is not mandatory.

Clinical picture :

1. External rotation and shortening of the limb.
2. Tenderness over the fracture site.
3. Inability of the patient to raise his leg.

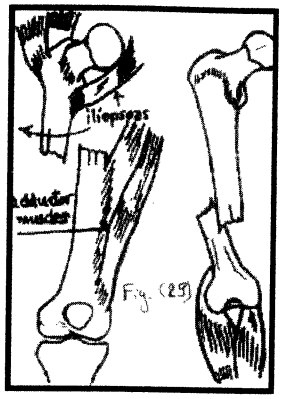
Treatment :

[A] In the elderly : internal fixation by plate & screws.
[B] In young persons :
1. Trochanteric fractures may be treated by immobilization and traction to the limb.
2. Subtrochanteric fractures are treated by internal fixation.

Complications of extracapsular fractures :

1. Thrombo-embolism.
2. Malunion leading to shortening, adduction and external rotation.

Fracture of the shaft of femur



Trauma :

Incidence : Can occur in children and adults & Associated injuries are common.
- May be direct as birth injury or car accident or
- Indirect as fall from a height.

Classification : The fracture may be :
1. Simple crack.
2. Spiral.
3. Transverse.
4. Two transverse fractures with a separate segment in between.

Morbid anatomy :

[A] Fractures at the upper third : The proximal fragment is flexed by the iliopsoas, abducted by the gluteal muscles & externally by the external rotators. The lower fragment is adducted by the adductor muscles, drawn proximally by the hamstrings & externally rotated by the weight of the limb.
[B] Fractures of the lower two thirds : The distal fragment is posteriorly angulated.

Treatment :

- **Certain principles should be followed :**

1. Any malunion will lead to osteoarthrosis of the knee joint.
2. Shortening more than 2 cm should not be allowed.

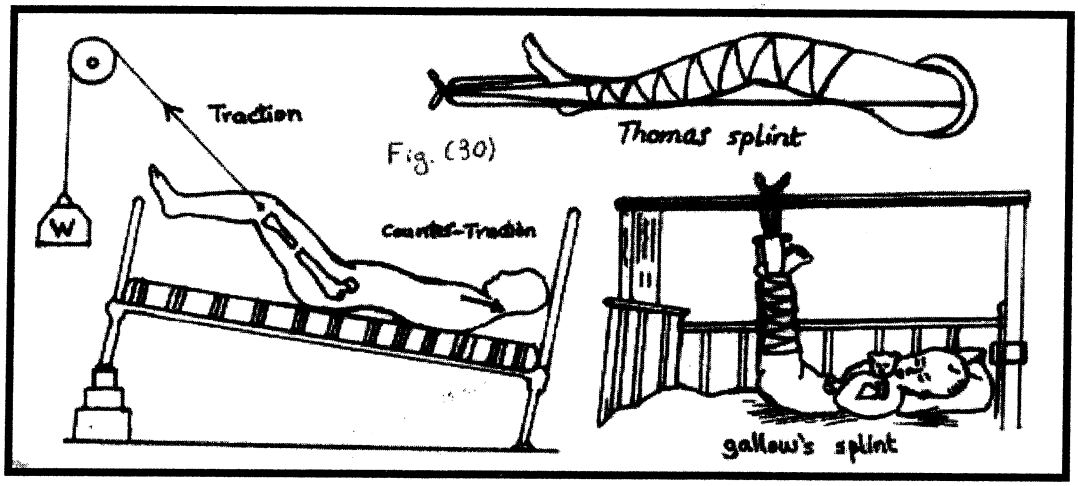
[A] Conservative treatment :

1. Sliding traction : Traction is applied to the distal fragment by weights and pulleys attached to the limb either by skin strapping in children or by skeletal traction in adults by inserting special pins in the upper end of the tibia.
- Counter-traction depends upon the weight of the body acting by elevation of the foot of the bed.
- In children below 2 years **gallow splints** are used by strapping of the legs to a cross piece, so that the pelvis is lifted from the bed.

2. Fixed traction : by using a Thomas splint.

- **Disadvantages of conservative treatment :**

1. Prolonged immobilization.
2. Liability to stiffness of the knee.
3. Difficulty in adjusting the ideal traction; excess traction may lead to distraction.
4. The lateral popliteal nerve may be injured by compression by Thomas splint.



[B] Operative treatment : (open reduction and internal fixation)

- This avoids the disadvantages of conservative treatment.

Indications :

1. Inability to perform closed reduction due to interposition of soft tissues.
2. Associated vascular injury.
3. Fractures of the proximal shaft as proximal segment can't be immobilized by conservative measures.
4. Double-level fractures.

Methods of open reduction and internal fixation :

- Fractures of the proximal shaft are fixed by plate.
- Fractures of the middle two quarters are immobilized by an intramedullary nail.

Complications of fracture of the shaft of femur :

1. **Vascular injury :** Displaced fractures of the lower third may injure the femoral or popliteal vessels.
2. **Nerve injury :** The lateral popliteal nerve may be injured by the outer bar of a Thomas splint.
3. **Non union** is uncommon.
4. **Malunion :** The usual deformities include shortening, varus and external rotation.
5. **Knee stiffness :** A serious complication.

Fractures of the Distal Femur

I- Supracondylar fracture

This fracture is very difficult to treat conservatively as the gastrocnemius muscle flexes the distal fragment, producing posterior angulation at the fracture line.

Treatment :

1. The best results are obtained by internal fixation of the fragments using plate and screw.
2. Conservative treatment is possible. **Two Steinmann's pins** are used; one is inserted in the tibial tubercle to provide traction of the distal fragment in the longitudinal axis of the femur, the other pin inserted in the distal fracture segment to provide forward traction at a right angle to the femur.

II- Intracondylar fractures

Mechanism of injury :

1. Indirect trauma. A fall from a height.
2. Direct trauma due to a blow to the anterior aspect of the flexed knee.

- **These fractures take the shape of T or Y fractures, which are similar to those occurring in the distal humerus.**

Treatment :

- This fracture is liable to be followed by osteoarthrosis of the knee. Every attempt should be made to reconstruct the joint surface especially in young patients.
- Open reduction and internal fixation by screws is the best line of treatment in young patients.

Intramedullary nail fixation :

Indication :

- Simple fracture of shaft of femur in an adult or elderly.
- Sometimes it is used for internal fixation of pathological fracture.

Contra-indications :

1. Compound fracture (risk of infection).
2. Comminuted fracture.
3. Fracture of the lower end of femur.
4. Presence of skin infection.

Technique of internal fixation by nail :

- An incision is done starting from ASIS to lateral border of the patella and the rectus femoris is retracted medially exposing the fracture.
- The two fragments are hold by bone holding forceps. The upper fragment is raised out of the wound and the nail is hammered towards the greater trochanter.
- When the nail reaches the greater trochanter the skin is incised and the nail is hammered more till its 1/3 comes out of the greater trochanter.
- Then the two fragments are reduced and fixed by the bone holding forceps.
- The nail is re-hammered back towards the knee.
- After six months the nail can be removed but if non-union occurs and when the nail is formed of vitalium it can be left.

Complications of intra medullary nail :

1. If the nail is too long it will reach the knee joint.
2. If the nail is too short the lower fragment will not be properly fixed.
3. If the nail is too thin, the bone fragments are going to rotate.
4. If the nail is too thick, it either becomes impacted or breaks the femur.
5. Fat embolism.
6. Osteomyelitis.
7. The nail must be of inert material.

Extensor Apparatus Injuries

Rupture above the patella : Occurs in elderly or on long term corticosteroid therapy.
Rupture below the patella : This occur in young people. Operative repair is necessary.

Fractures of the patella

Mechanism of injury :

1. Indirect injury : due to forced flexion of the knee when the quadriceps muscle is contracting. In this case the fracture is transverse.
2. Direct injury : due to trauma to the anterior aspect of the flexed knee leading to comminuted fractures.

Types of fracture :

[A] Undisplaced transverse fractures :

Clinically :

- Pain and tenderness at the site of the fracture.
- Active extension of the knee is still possible although painful.

Treatment : Immobilization of the knee in plaster for 6 weeks.

[B] Displaced transverse fractures :

- The proximal segment of the patella is drawn upwards by the quadriceps muscle.

Clinically : A gap is palpable in the patella and active extension of the knee is impossible.
Treatment : Is by open reduction and internal fixation.

[C] Comminuted fractures of the patella :

Treatment : Patellectomy followed by reconstruction of the soft tissue component of the extensor apparatus.

[D] Upper or lower polar fracture :

1. If undisplaced, immobilization for 6 weeks is satisfactory.
2. If displaced, open reduction and internal fixation.
3. Very small fragment can be excised and the extensor mechanism is repaired.

[E] Vertical fractures : Can be treated conservatively.

Complications of fractures of the patella :

1. Secondary osteoarthrosis.
2. Chondromalacia patellae.
3. Stiffness of the knee.

Injuries of the knee joint

Meniscus Injury

The meniscus has an important role in :

1. Increasing the stability of the knee.
2. Control the complex rolling and gliding actions of the joint.
3. Distributing load during movement.

The medial compartment of the knee carries about 90% of the load during weight bearing & the medial meniscus is much less mobile than the lateral, partially because of its attachment to the capsule. Accordingly, the meniscal lesions are more common on the medial side than on the lateral. The meniscus is split in its length by a force crushing it between the femur & tibia. It commonly affects football players when weight is being taken on the flexed knee & there is a twisting strain.

Pathological anatomy :

The tear is usually longitudinal **(bucket-handle tear)** but may be transverse **(parrot-beak tear).**
A meniscus is avascular and a tear does not heal unless it is peripheral (outer third) which is vascularized from the capsule.

Clinical features :

There is a typical history of injury while playing a game. The player falls, feeling pain in the knee. He cannot resume the game, but can walk home with a limp. The patient may complain of giving way, locking of the joint or recurrent effusion.

Examination reveals :

1. Knee effusion.
2. Limitation of complete extension when the knee is locked.
3. Tenderness localized to the joint line on the medial side.
4. Positive Mc Murry's sign (a click may be elicited when the knee is rotated in certain degree of flexion).
5. Atrophy of the quadriceps muscle is noticed in long standing cases.

Investigations :

1. Plain X-rays are normal.
2. MRI is the most reliable method & may even reveal tears that may be missed by arthroscopy.
3. Arthroscopy has the advantage that if a lesion is identified, it can be treated at the same time.

Differential diagnosis :

1. Loose bodies.
2. Recurrent dislocation of the patella.
3. Fracture of the tibial spine.
4. A partial tear of the medial collateral ligament.
5. A torn anterior cruciate ligament.

Treatment :

Locked knee :

Usually the knee unlocks spontaneously. If it is does not unlock or if attempts to unlock cause severe pain, arthroscopy is indicated and if the tear is confirmed, the offending fragments are removed.

Conservative treatment :
Often acceptable if the knee is not locked. The joint is held straight in a posterior slab for 3 - 4 weeks; quadriceps exercises are encouraged.

Operative treatment is indicated if :
1. The joint cannot be unlocked.
2. Symptoms are recurrent.

- After arthroscopic or open meniscectomy, a postoperative hemarthrosis may need aspiration. Post-operative pain and stiffness are reduced by prophylactic non-steroidal anti inflammatory drugs. Quadriceps strengthening exercises are important.

Fractures of the tibia

These are classified into :
1. Fractures of the proximal tibia.
2. Fractures of the shaft.
3. Fractures of the distal tibia (ankle fractures).

Fracture of the Proximal Tibia

Mechanism of injury :
A fall from a height or a direct blow may fracture one tibial condyle or both.

Clinical features :
The knee is swollen and may be deformed. Bruising is usually extensive.

Treatment :
1. Traction through the tibia 7cm below the fracture.
2. Plaster fixation after closed reduction. Advantage : The patient does not need to remain in hospital.
3. Open reduction and internal fixation if closed reduction fails.

Fractures of the Shaft of the Tibia

May be :
[A] Isolated fracture of the tibia : usually due to torsion leading to a spiral fracture.
Treatment : Usually conservative by an above-knee plaster cast.

[B] Fractures of the tibia and fibula :
Trauma :
1. Direct trauma e.g. road traffic accidents.

- The fractures occur at the same level in the two bones and are transverse or oblique.
- They are usually compound and there is often significant displacement.

2. Indirect trauma e.g. due to a torsion or bending injury, as in skiing & football players. Both injuries may be compound from within.

Treatment :

Method	Indication
1. Closed reduction and plaster fixation	Stable reduction can be obtained.
2. Skeletal traction through the lower end of tibia or calcaneus	If stable reduction cannot be maintained by plaster alone.
3. External skeletal fixation	In compound fractures of the tibia as it allows for proper reduction and at the same time leaves the wound exposed for dressings.
4. Internal fixation with plate & screws or intramedullary nail	In multiple fractures or in patients in whom reduction cannot be obtained by closed methods.

Complications of fractures of the tibia :

1. Skin damage : as the shaft of the tibia is directly subcutaneous, fractures of the tibia are usually compound.
2. Vascular injuries : the distal end of the popliteal artery.

3. Delayed and non-union : This occurs in up to 20% of fractures of the tibial shaft due to:
(a) Significant initial injury.
(b) Soft tissue damage. Early skin cover is vital for healing of the fracture.
(c) Infection.
(d) Comminution.
(e) Inadequate immobilization.
(f) Distraction.
Treatment : Internal fixation and cancellous bone grafting.

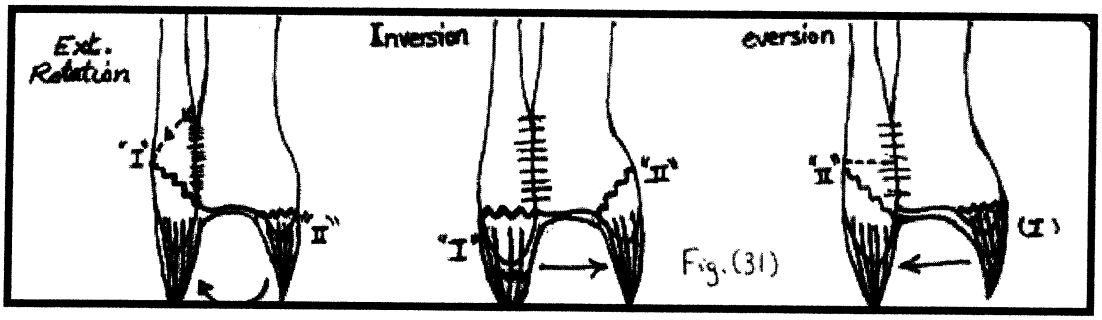
4. Malunion : may occur after poor closed reduction.
5. Infection : Serious complication
6. Stiffness : of the ankle or subtalar joint.

Ankle fracture

Causative trauma :

1. External rotation trauma.
2. Severe adduction with inversion.
3. Abduction with eversion.
4. Vertical compression e.g. Fall from a height.

Although the mechanism is described as if the foot moves on a fixed tibia, in practice, these injuries nearly always occur with the foot fixed, e.g. by a hole in the ground , while the tibia continues to move, driven by the body weight.



These fracture are classified according to the mechanism of injury :

1. External rotation injury : (Pott's fracture)

This is the commonest type.
Stage I: The talus pushes the lateral malleolus posteriorly producing an oblique fracture of the distal fibula with a characteristic fracture line which runs downwards and forwards to the level of the ankle joint. The ankle is stable.
Stage II: Rupture of the deltoid ligament, or an avulsion fracture of the medial malleolus. Now, the ankle is not stable. Lateral dislocation of the talus may occur.
Stage III: The posterior rim of lower tibia is fractured (fracture third malleouls).

- **Now, further posterior dislocation of the talus will occur.**

2. Inversion (adduction) injuries :

Stage I: Rupture of the lateral ligament or an avulsion fracture of the lateral malleolus, the ankle is stable. **(Sprains & ruptures of the lateral ligament are the commonest ankle injuries).**
Stage II: Next, fracture of the medial malleolus occurs with a near vertical fracture line. Medial displacement of the unstable ankle occurs.
Stage III: Rarely, fracture of the third malleolus occurs with further posterior displacement.

3. Eversion (abduction) injuries :

Stage I: Rupture of the deltoid ligament or transverse fracture of the medial malleolus. The ankle is stable.
Stage II: Oblique fracture of the lower fibula extending upwards and outwards from the level of the ankle, lateral displacement then occurs.
Stage III: Rarely, fracture of the third malleolus with further posterior displacement.

4. Vertical compression injuries :

The pattern o the fracture depends on the position of the ankle at the time of injury.
(a) Burst fracture of the ankle : The foot is neutral at time of injury. The distal end of the tibia is comminuted and the talus is driven proximally between the tibia and fibula.
(b) Anterior marginal fracture of the tibia : The ankle is dorsiflexed at the time of injury. There is anterior sublaxation of the talus.
(c) Posterior marginal fracture of the tibia : The ankle is plantar-flexed at the time of injury

5. Internal rotation injuries :

These are rare and produce injuries similar to those of inversion.

Clinical picture :

All the signs of a fracture are present. **Swelling** is particularly severe and **deformity** is evident which is variable according to the type of fracture.

Investigation :

Stress X-rays are very important for detecting ligamentous injuries.

Treatment :

The aim is to restore :

1. The anatomical position of the talus.
2. The joint line parallel to the ground.
3. The smooth articular surface.
4. Stability until the fracture(s) unites by plaster cast or internal fixation.

Guidelines :

1. Stable fractures :
- Should be treated by application of a weight bearing below-knee plaster cast for 6 weeks.

2. Unstable undisplaced fractures :
- Should be treated by application of non weight-bering plaster in 6 week.

3. Unstable displaced fractures :
- In young patients : Accurate open reduction and internal fixation.
- In elderly patients : Closed reduction and application of a below-knee plaster cat for 6 weeks.

Complications :

1. Malunion results from failure to reduce and stabilize unstable fractures.
2. Non-union occasionally occurs in fractures of the medial malleolus. For this reason, this fracture is always best treated by internal fixation.
3. Secondary degenerative osteoarthrosis.
4. Ankle stiffness.

Deformities of the bones & joints

Some important definitions

Words used to describe parts		
Cubitus = Elbow	**Coxa** = hip	**Genu** = knee
Talipes = heel and foot	**Pes** = forefoot alone	

Words used to describe deformities	
Valgus	A deformity where the part is directed away from the midline of the body.
Varus	A deformity where the part is directed towards the midline.
Equinus	Vertical position of the tarsus as a horse's foot.
Calcaneus	Deformities where the calcaneum assumes a more vertical position than normal.

Words used to describe operative procedures	
Osteotomy	Division of a bone.
Tenotomy	Division of a tendon.
Arthrodesis	Fusion of a joint.
Tenodesis	Deliberate anchoring of a tendon performed to control abnormal movement.
Arthroplasty	Reconstruction of a joint aimed at restoring movement.
Pseudoarthrosis	Describes a false joint which may be deliberately sought as in certain operations on the hip or results from a pathological process as when a false joint forms at the site of an ununited fracture.

- Normally the forearm is in a position of slight abduction with the arm constituting an angle of about 10 - 15° known as the carrying angle. In cubitus valgus, this angle is increased so that the hand deviates away from the body while in cubitus varus the reverse occurs.
- The importance of valgus deformity is the liability for delayed ulnar palsy to develop. The nerve should be transported to the front of the elbow.

Deformities of the hip

Congenital dislocation of the hip

Incidence :

- 1 in 1000 newborn babies.
- More common in girls than in boys.
- The left hip is more often affected than the right. In 1/3 of cases both hips are affected.

Etiology :

1. There is a familial tendency. 2. Intrauterine malposition. 3. Postnatal posture.

Pathology : The acetabulum is unusually shallow. The femoral head slides out posteriorly and then upwards. The joint capsule is stretched.

Clinical features:

1. Before the baby starts walking, an observant mother may spot **asymmetry, a clicking hip** or **difficulty in applying the napkins** because of limited abduction.
2. After walking starts, **asymmetry is more obvious** and now **a limp** also becomes apparent.

Examination :

[A] Unilateral dislocation	[B] Bilateral dislocation
1. The skin creases lie at different levels on the two sides. 1. The affected leg is slightly short & rotated externally. 3. Trendlenburg's test is always positive.	1. More difficult to detect because there is no asymmetry. 2. The perineal gap is abnormally wide and abduction may be limited.

Special tests :

- Congenital dislocation should be diagnosed soon after birth where treatment is simple & most effective.
- Routine postnatal examination must include special tests for instability.

Ortolani's test :

- The baby's thigh is held with the middle finger behind the greater trochanter and the thumb in front, during abduction the greater trochanter is pressed forwards and during adduction the femoral head is pushed gently backwards.
- The test is positive if there is a click during the abduction manoeuvre indicating that the tip has been reduced.

Barlow's test :

- If the hip is already reduced, the adduction manoeuvre will allow the femoral head to be gently levered out of the socket **(Barlow's modification of Ortolani's sign).**

Investigation :

1. X-ray examination : of little value in the newborn because the head of the femur is entirely cartilaginous and not visible radiologically.

2. Ultrasound scanning : can visualize the shape of the acetabulum and the position of the femoral head with reasonable accuracy.

- **In a child of one year age or more :** Plain X-ray shows that the acetabular socket is unusually shallow.

Treatment :

1. In the newborn : Wait 3 weeks, if the hip is still unstable, it is reduced and splinted in abduction **(Von Rosen splint).** This is maintained for 3 - 6 months.

2. Between the age of 6 months to 6 years : The objectives are to reduce the hip and hold it reduced until acetabular development is satisfactory.

(a) Gradual closed reduction is applied to both legs and abduction is gradually increased until by 3 weeks & Splintage for about 12 months.

(b) Operative reduction.

3. After the age of 6 years :

- Unilateral dislocation : Operative reduction.
- Bilateral dislocation : The deformity and waddling gait is symmetrical and the risk of operative intervention is greater because failure on one or other side turns this into an asymmetrical deformity. Therefore, in these cases most surgeons would avoid operation.

Deformities of the Foot

Congenital talipes equinovarus (club foot) :

- It is the commonest variety of talipes. Bilateral in one third of cases.

Etiology : Theories :

1. The lower limbs of the fetus are forced against the wall of the uterus.
2. Ischemic contracture of the soft tissues of the foot.
3. Developmental defect of the soft tissues of the legs.

Pathological anatomy :

1. Planter flexion of the foot at the ankle.
2. Inversion of the calcaneus and of the navicular on the talus.
3. Adduction of the bones of the forefoot at the mid tarsal and tarso-metatarsal joints.
4. Shortening of all ligamentous structures on the medial side of the foot. Shortening of the flexors of the foot and toes.

Clinical features :

1. Deformity. 2. Painful callosities develop years later.

- **Gentle attempts at passive correction show the deformity to be fixed.**
- **Although the feet of a normal baby may lie naturally in an equino-varus position, they can be passively dorsiflexed until the toes touch the front of the leg.**

Differential diagnosis :

Paralytic talipes equino-varus : Usually unilateral and may be due to poliomyelitis, at first the deformity can be corrected by manipulation but later the deformity becomes rigid & permanent.

Treatment :

1. Stretching and splinting : Treatment begins within 2 or 3 days of birth. The process is repeated weekly for 6 - 8 weeks until the foot is overcorrected. Finally correction must be confirmed by X-ray.

2. Operation : In resistant cases. The tendo Achillis is lengthened and the tight invertors and plantar flexors are elongated or divided. The foot must be corrected fully and held in plaster.

3. Treatment after correction : Splintage continues until the child starts walking, thereafter, correction can be maintained by **Denis Browne night splints and special shoes.**

4. Later operations :

(a) A less than 5 years old, open division of the contracted soft tissues.
(b) Over the age of 5 years, correction of deformities is impossible without bone reshaping.
(c) Over the age of 10 years, lateral wedge tarsectomy or triple arthrodesis.

Flat foot (Pes plannus)

- At birth the foot is flat and the normal arch is only acquired when the infant stands.
- The arch is maintained in adult life by the shape of the bones of the foot, their connecting ligaments and the action of the muscles of the calf and sole.

There are 4 main varieties of flat foot :

1. Infantile : This is physiological.

2. Spasmodic flat foot :

- Uncommon painful condition which presents insidiously in adolescence.
- The foot is everted and hence the medial arch appears flat.
- The tendons of the peroneal & long toes extensors can be seen in continuous contraction.
- Attempts to passively invert the foot are resisted by these muscles and are painful.
- Treatment is usually unsatisfactory.

3. Idiopathic adult flat foot :

- The commonest form of symptomatic flat foot in adults.
- Repeated episodes of foot strain.
- The condition is gradually progressive and presents with aching pain in the foot after long continued standing.
- The medial longitudinal arch gradually collapses causing pain centred in the medial part of the sole.
- Pain is provoked if these ligaments are stressed by dorsiflexing the forefoot.
- In later stages, the foot becomes painless and the gait is inelastic. The foot is rigid & flat.

Treatment :

- Indicated if the flat foot is symptomatic.
- The pain of foot strain is relieved by rest.
- Its prevention may require arch supports.
- Exercises to improve the function of the intrinsic muscles of the sole and of the toe flexors may prevent further deterioration.
- When the foot has finally collapsed, little can be done to improve its function and the patient should be advised to accommodate his life to his feet by taking a sedentary job.

4. Traumatic flat foot : This is caused by fractures which abolish the longitudinal arch.

Bone infection

Infection may reach the bone and joints via the blood stream from a distant site, or by direct invasion from a skin puncture, operation or an open fracture.

Acute Hematogenous Osteomyelitis

Acute osteomyelitis is almost a disease of children. (Males are more affected than females, as they are more liable to trauma).

Etiology :

- **Causative organisms :** Usually Staphylococcus aureus (80%). Less often Streptococcus pyogenes or Gram negative Hemophilus influenza.
- **Source :** septic focus e.g. Boils or IV lines.
- **Route :** the blood

Pathology :

- **Site :**

1. In adults, hematogenous infection is more common in the vertebrae (especially thoraco lumbar).
2. In children, the lesion is almost always in the **metaphysis of long bones**, most often at the lower end of femur, upper end of tibia.

- **This predilection for the metaphysis is due to :**

1. The peculiar arrangement of blood vessels (supplied by end arteries).
2. Most liable to trauma.
3. It is the actively growing end of bone.
4. Attachment of ligaments and muscles to it (subjected to strain).

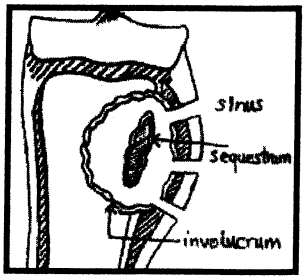
Natural history of osteomyelitis :

1. Inflammation and suppuration :
There is congestion and exudation. By the second or third day, pus forms within the bone causing intense pain and obstruction of blood flow.

2. Extension and bone necrosis (Sequestration) : The extension may be:
(a) Horizontal spread : Results in the formation of subperiosteal abscess. Later, the abscess may open on the skin with resultant discharging sinuses.
(b) Vertical extension : Along the medulla may result in thrombosis of the major nutrient vessels and massive sequestration of the shaft.

- **The sequestrum** is smooth, shiny white, and devoid from periosteum and so gives a metallic sound on percussion by a probe. Separation of a sequestrum is known by probing (it moves) and by X-ray where a black hallow is seen surrounding the sequestrum which is dense.

3. New bone formation in two sites :
(a) The deep layers of the stripped periosteum by the end of 2nd week.
With time the new bone thickens to form an **involucrum** enclosing the sequestra.
If the infection persists, pus may continue to discharge through perforations **(cloaca)** in the involucrum and sinuses to the skin surfaces,
the condition is now established as a chronic osteomyelitis.
(b) Around the Haversian canals resulting in bony sclerosis.



4. Resolution :
The bone around the zone of infection is at first osteoporotic, later there is sclerosis and thickening of the bone. Remodelling may restore the normal contour.

Complications of acute osteomyelitis :

Systemic : Metastatic infection (pyemic abscesses).

Local :

1.Suppurative arthritis.
2. Altered bone growth.
3. Chronic osteomyelitis.
4. Pathological fracture.

Clinical features :

1.Type of patient : **usually child**
2. Severe limb **pain, malaise** and **fever**.
3. The earliest sign is severe **localized tenderness** over the inflamed part of the bone.
4. Later, there is **warmth, edema** and the skin is **red.**
5. Although any **movement** of the limb is **painful, the neighbouring joint can be moved passively and there may be an associated effusion.**

Investigations :

1. Laboratory : Leucocytosis, raised ESR and blood culture may be positive.
2. X-ray : Is normal during the first two weeks, then rarefaction and periosteal new bone formation appear. **Treatment should not be delayed while waiting for these to appear.**
3. Radioscintigraphy : With ^{99m}Tc-HDP (hydroxymethylene diphosphonate) reveals increased activity in the very early stage.
4. The most certain way to confirm the clinical diagnosis is **aspiration of pus.**
5. MRI : can detect osteomyelitis before the appearance of radiological changes in plain radiology.

Differential diagnosis :

1. Cellulitis .
2. Acute suppurative arthritis. Tenderness is over the joint and all movements are painful & limited.
3. Rheumatic arthritis.
4. Hemarthrosis.
5. Ewing's sarcoma.

Treatment :

1. Supportive treatment : for pain and dehydration.
2. Splintage : of the affected part.

3. Antibiotic therapy :

The core of treatment : is early administration of high-dose IV antibiotics that are effective against staphylococci. Start with flucloxacillin intravenously or second generation cephalosporin & gentamycin for 3 or 4 days, then once the condition begins to improve, oral antibiotics are prescribed for another 3-6 weeks.

4. Surgical drainage :

Indication : If the patient is first seen after 48 hours or if there is no good response to antibiotics within 2 days

Method :

- Done under general anaesthesia and under a tourniquet.
- The inflamed area is explored; the periosteum is opened to evacuate the pus which is sent for culture and sensitivity.
- Some surgeons advise drilling of the cortex to evacuate any pus in the medullary cavity.

After care :

1. Splintage is continued until the inflammation subsides and the X-ray shows that there is no risk of a pathological fracture.
2. Full weight bearing is usually possible after 3 - 4 weeks.

Chronic bone infections

Chronic non specific osteomyelitis	Chronic specific osteomyelitis
1. Chronic osteomyelitis as a sequel to acute osteomyelitis. 2. Brodie's abscess. 3. Chronic sclerosing osteomyelitis (Garre's type).	1. Tuberculosis. 2. Spirochetal infection (Syphilis). 3. Mycotic infection.

Chronic Non-Specific osteomyelitis

Chronic osteomyelitis as a sequel to acute osteomyelitis

The causative organisms :

- Usually a mixed infection (Staphylococcus aureus, E. coli, Strept. pyogenes, Proteus & Pseudomonas).
- Nowadays, chronic osteomyelitis more frequently follows an open fracture or operation, in the presence of foreign body (implants). The causative organism is usually Staph. epidermidis which is normally non pathogenic .

Pathology :

One or more of the following factors are present and maintain chronicity :

1. Bone cavity surrounded by dense sclerosis.
2. Sequestrum which acts as an irritant and harbours bacteria.
3. Bacteria in the fibrous tissue where they remain dormant, and may be activated at any time.
4. Sinuses which lead to the skin surface favouring secondary infection.

Complications :

1. Acute exacerbation.
2. Pathological fracture.
3. Toxemia and amyloidosis.
4. Retardation or arrest of growth.

Clinical picture :

1. History of acute osteomyelitis may be given.
2. A sinus which is discharging pus and sometimes small pieces of sequestra **(the commonest presentation).**
3. Pain and swelling in the affected bone.
4. There may be atrophy of the surrounding muscles.
5. On examination, the affected bone is thickened, tender with sinuses that are lined by red granulation tissues, exuding pus and when the sinus is probed, the probe will reach the bone.

Investigations :

1. Plain X-ray (the most important) : The bone is thickened with patchy and irregular sclerosis surrounding a bone cavity that may contain sequestra.
2. Radioisotope scanning: Using ^{99m}Tc-HDP scans : useful for showing up hidden foci of infection.
3. CT and MRI : Are valuable in planning operative treatment.
During acute flares the ESR and white cell count will be increased.

Treatment :

- Surgery for removal of dead bone and drainage of the cavity is the main line of treatment.
- Antibiotics alone are of little value because they cannot reach the bacteria hidden in avascular tissue & sequestra. They are of benefit in an acute exacerbation & when given with operative drainage.

Operative procedure :

1. Saucerization of a bone cavity.
2. Sequestrectomy.
3. Excision of the sinus tract.
4. Obliteration of the dead space by cancellous bone chips or local muscular flaps.

Brodie's Abscess

It affects patients with high resistance resulting in localization of infection caused by Staph. aureus or albus.

Pathology :

A chronic bone abscess situated in the metaphysis of long bone, commonly the tibia (upper end) surrounded by dense sclerosis.

X-ray : There is a translucent area surrounded by sclerosis.

Treatment : Under antibiotic coverage the cavity is deroofed.

Chronic Sclerosing Osteomyelitis (Garre's Type)

- This is a non suppurative type of osteomyelitis affecting shafts of long bones.
- There is diffuse thickening of bone with encroachment on the medullary canal (no pathological fracture).
- There is no associated sequestration or sinus formation.

Clinical features:

- Pain may be severe and is exaggerated by night.
- The condition is to be differentiated from syphilitic osteomyelitis and Ewing's sarcoma.

Treatment:

Guttering of the cortex is needed in order to release the tension in the medullary canal.

Tuberculous infection of Bone

In contrast to pyogenic infection, tuberculosis causes a granulomatous reaction which is associated with tissue necrosis and caseation.

Pathology :

The organism is always carried by blood stream form a primary focus elsewhere in the body (i.e. it is secondary tuberculosis).

The special features of T.B osteomyelitis are :

1. Bone destruction and rarefaction.
2. Caseation results in cold abscess and sinus formation.
3. Healing occurs by fibrosis.
4. Sequestration is unusual and if present the sequestra are small.
5. Tuberculosis can destroy the epiphyseal cartilage plate and involve the neighbouring joint (unlike pyogenic osteomyelitis).

- Depending on virulence of the organism and patient resistance will end in one of the following types :

1. **The encysted type (Brodie's abscess).**
2. **The infiltrating type (the commonest type).** A good length of bone is involved with spread to the nearby joint.
3. **Atrophic type (caries sicca).**
4. **Hypertrophic type (excessive new bone formation),** e.g. tuberculous dactylitis.

Complications :

1. Cold abscess formation.
2. Involvement of a joint.
3. Sinus formation.
4. General tuberculous toxaemia, miliary TB and amyloid disease.

Clinical features :

1. **Tuberculous osteomyelitis** is usually a disease of children.
2. **T.B toxaemia.**
3. **Local manifestations near the joint :**
   - Pain of insidous onset, aching in nature and more at night.
   - Swelling near the joint with marked muscle atrophy.
4. **There may be a cold abscess or sinus formation.**

Investigations :

1. Lab : ESR is elevated, blood picture shows anemia, leucopenia & relative lymphocytosis. Tuberculin test is positive.

2. X-ray : There is localized bone destruction with widespread rarefaction with no new bone formation (except in hypertrophic type).

Treatment :

1. General measures :

Adequate diet, iron supplement or even blood transfusion and isolation of the patient (If there is associated pulmonary tuberculosis with positive sputum).

2. Antituberculous drugs :

Triple therapy consisting of :
Rifampicin 10 mg/kg wt, Isoniazid 5 mg/kg wt and Ethambutol 25 mg/kg wt/day are given orally for at least 2 to 3 months and thereafter Rifampicin and Isoniazid for a further 6 -12 months.

3. Immobilization :

Fixation of the limb in position of function in a splint or plaster for up to 6 months till clinical, laboratory and radiological evidence of healing occur.

4. Operative treatment :

Indications :
- Cold abscess.
- Failure of conservative treatment

Methods :
- Operative drainage and curettage are to be performed.
- In certain sites like tuberculosis of the rib, resection of the rib may be performed.

Other Forms of Osseous Tuberculosis :

[1] Tuberculosis of flat bones (periostitis) :

The surface of the bone (ribs, sternum, scapula or facial bones) is eroded & abscess forms which often opens on the surface with sinus formation.

[2] Tuberculosis of short long bones (TB dactylitis) :

- Metatarsals, metacarpals and phalanges are involved.
- The infection starts in the diaphysis. There is central destruction, and the periosteum is raised by tuberculous granulation tissues, with subperiosteal new bone formation. The bone thus becomes fusiform in shape **(spina ventosa).**

Septic Arthritis

Etiology :

- **Age :** Common in children.
- **Organism :** Staph. aureus. In children below 3 years of age, it is mainly due to Hemophilus influenza .
- **Routes of infection :**

1. Following joint punctures.
2. Direct extension from a nearby infection such as osteomyelitis.
3. By hematogenous spread from a distant site.

Pathology :

1. The synovial membrane (synovitis) : Becomes red, swollen and exudes pus.
2. Articular cartilage destruction with exposure of the bone ends.
3. Pus may burst out of the joint to form abscesses and sinuses.
4. Later with healing, the raw articular surfaces may adhere, producing **fibrous or bony ankylosis.**
Recovery of joint mobility can be achieved if treatment begins early at the stage of synovitis before damage of the articular cartilage.

Complications :

1. Chronicity.
2. Pathological dislocation.
3. Pyemia.
4. Secondary osteoarthrosis.

Clinical features :

- General manifestations of **toxemia.**
- **Severe throbbing pain and swelling** of the affected joint.
- There is **deformity, redness, tenderness & limitation of joint movements** equally in all directions.
- There is **muscle spasm.**

Investigations :

1. Lab : High Leucocytosis and elevated ESR
2. Aspiration of the joint : Reveals pus.
3. X-ray :

- In the early cases (first 2 - 3 weeks), no radiological abnormalities could be detected.
- Later, there is rarefaction of the articular ends of the bones followed by narrowing of joint space & irregularity of joint surfaces and subchondral sclerosis.

Differential diagnosis :

1. Acute osteomyelitis.
2. Rheumatic fever.
3. Hemarthrosis : There is a history of trauma and aspiration reveals blood.
4. Tuberculous arthritis : The onset is insidious and is chronic. A cold abscess may be present. Muscle atrophy is marked and X-ray shows rarefaction of bone ends.

Treatment :

1. Systemic antibiotics.
2. Prompt drainage :

- Drainage is either arthroscopic or by open **arthrotomy.**

3. The joint must be rested by splintage.
4. If the articular cartilage has been preserved, gentle and gradually increasing active movements are encouraged. If the articular cartilage has been destroyed, the joint is immobilized in the optimum position of function until ankylosis occurs.

Tuberculosis of Joints

- Joints commonly affected are the hip and knee; but no joint is immune.
- Infection reaches the joint by :

1. Hematogenous spread from lung.
2. Direct spread from a focus in the adjacent bone .

Pathology :

- **The site of initial lesion :**

1. Synovial (more common in children and in the knee).
2. Osseous where a bone focus may open into the joint.

- Whatever the site of initial lesion, in time, all joint components become affected in the untreated case.
- **The synovial membrane :** Thickened with numerous tubercles which may form a caseous mass.
- **Subchondral bone :** Is invaded by granulation tissue.
- **There is marked osteoporosis and bone destruction** without new bone formation.
- **The surrounding muscles** undergo marked atrophy.
- **Sequelae :** It always proceeds to complete destruction of the joint with any of the following sequelae:

1. Healing with fibrous ankylosis.
2. Cold abscess formation.
3. Pathological dislocation.
4. Generalized dissemination.
5. Amyloid disease.

Clinical Features : The patient is usually a child or young adult.

Symptoms :

- **General manifestations of tuberculosis** e.g. low grade night fever, loss of weight and anorexia.
- **Pain :** The earliest symptom that occurs only during exercise (night starts or cries).

Signs:

- **Swelling :** Results from thickening of synovial membrane and rarely effusion.
- **Muscle wasting** is characteristic.
- **Movements are limited in all direction.**
- As articular erosion progresses **the joint becomes stiff and severely deformed.**
- **Cold abscess and sinus formation :** In late cases.

Investigations :

[A] Laboratory investigations :

1. There is anemia, leucopenia with relative lymphocytosis, and elevated ESR.
2. Tuberculin test is positive.
3. Synovial fluid assessment reveals low or absent sugar, elevated white cell count. Bacteriological assessment may reveal tubercle bacilli.
4. Synovial membrane or regional lymph node biopsy.

[B] X-ray : Shows soft tissue swelling, rarefaction of the bone on both sides of the joint, narrowing of the joint space and blurring of the joint line from erosion of subchondral bone.

Treatment :

[A] Early cases :

1. Medical & general : See T.B bone.

- If started early, the joint may heal and function may be completely restored.

2. Local measures :

- Rest, traction, occasionally Splintage : should be continued for several months.

[B] Late cases :

If the articular surfaces are destroyed, the joint is immobilized until all signs of disease activity have disappeared, then the joint is arthrodesed in the position of maximum function .

Perth's Disease

Osteochondritis juveniles of the hip

- **Idiopathic** disease in children.
- **The femoral head** becomes ischemic, then softened and fragmented resulting in distortion of the head and neck of femur. Later bone repair takes place and the deformity is well-established.
- **The disease presents by limping.**
- **Treatment :** Is by traction for the acute phase to prevent deformities, and by corrective osteotomy in case the deformity is established.

Diaphyseal Aclasis (Hereditary Multiple Exostosis)

- **Characterized by :** The appearance of multiple exostosis at the metaphyses as the child grows.
- **Inherited as :** An autosomal dominant
- **Due to :** Failure of growth plate modelling. Each exostosis is covered by cartilaginous cap (osteochondromata), it grows only while the child grows and any enlargement after that may indicate malignant change to a chondrosarcoma.

Clinical features :

- Usually the only complaint of the patient is **multiple swellings.**
- **The child may be slightly short with mild or moderate bowing** of the arms or legs.
- **Picture of complications:** Pressure on a neighbouring nerve or blood vessels, or they may interfere with the movement of a joint.

Investigation :

X-ray : shows the pathognomonic exostosis as well as broadening of the metaphyses.

Treatment :

An outgrowth that is causing trouble or enlarges after the parent bone has stopped should be removed.

Paget's disease (Osteitis Deformans)

The condition can affect any bone in the body; the spine, the skull, the pelvis, the femur and the tibia are the most commonly involved bones.

Pathology :

- The pathological process begins with osteoclastic resorption of existing bone. This is followed by osteoblastic regeneration of a primitive highly vascular bone .
- Cortical bone becomes increasingly thick and less well demarcated from adjacent cancellous bone, and finally it assumes the mosaic pattern of lamellar bone.
- While resorption predominates, the bone is easily deformed. in the late osteoblastic stage, the bone becomes increasingly sclerotic and brittle.

Clinical features :

- The patient complains of limb **pain and headache.**
- **The skull enlarges** and larger hats are required.
- **Blindness** due to pressure on the optic nerve or **paraplegia** due to narrowing of the vertebral canal may occur .
- **Deformities** develop as kyphosis, coxa vara and bowing of the legs.

Complications :

1. **Nerve compression** (blindness, paraplegia, and deafness).
2. **Pathological fractures.**
3. **Osteoarthrosis** of the joint adjacent to deformed bone.
4. **Osteosarcoma.**
5. **High output heart failure** (due to A - V shunt within the bone).

Treatment :

Most patients with Paget's disease never have symptoms and require no treatment except when complications arise. Indeed, there is no specific therapy, but drugs such as calcitonin and diphosphonates can control the disease.

Charcot's Osteoarthropathy (Neuropathic Arthritis)

Pathology :

- This is a rapidly progressive degeneration in a joint which lacks deep sensations .
- The affected joint is severely disorganized after repeated minor injuries .
- In the lower limb it is associated with tabes dorsalis, cauda equina and peripheral neuropathy (DM), while in the upper limb the cause could be syringomyelia (the shoulder) or leprosy (the wrist).

Clinical features :

There is painless swelling and deformity of the joint. There is effusion, synovial thickening and loose bodies. The patient complains of weakness, instability and abnormal mobility can be elicited. Signs of accompanying neuropathy are present, as anesthesia, lost tendon jerks and perforating ulcers of the foot.

X-ray :

There is gross bone destruction, widening of joint spaces and the joint is often sublaxated or dislocated. There are irregular calcified masses in the capsule.

Treatment :

- The underlying condition may need treatment, but the affected joints cannot recover. They should, if possible, be stabilized by external splintage. Surgery is not advised.
- Amputation may be indicated in Charcot's joint of the feet.

Bone tumors

General principles :

Classification of primary bone tumors

Cell type	Benign	Malignant
Bone	Ivory osteoma Osteoid osteoma Osteoblastoma	Osteosarcoma
Cartilage	Chondroma Osteochondroma Chondroblastoma Chondromyxoid fibroma	Chondrosarcoma
Fibrous tissue	Non ossifying fibroma	Fibrosarcoma
Marrow	Eosinophilic granuloma	Ewing's sarcoma Myeloma Reticulum cell sarcoma
Vascular	Hemangioma	Angiosarcoma
Uncertain	Giant cell tumor	Malignant giant cell tumor

Classification of bone tumors according to their site

Vertebral lesions	Flat bones	Long bones
Osteoid osteoma Osteoblastoma Hemangioma Myeloma Secondaries	Secondaries Myeloma Chondrosarcoma	1. Epiphysis : • Giant cell tumour • Chondrosarcoma 2. Metaphysis : Osteosarcoma 3. Diaphysis : • Secondaries • Ewing's sarcoma

Differential diagnosis of the bone tumours on radiography :

1. Secondary tumours.
2. Osteomyelitis.
3. Callus from unnoticed stress fracture.
4. Simple bone cyst.
5. Brown tumour of hyperparathyroidism.
6. Myositis ossificans.

Biopsy :

It is essential to establish the diagnosis. The edge of the tumor is the ideal part to biopsy because :

1. The center may be necrotic.
2. To obtain tumor and normal surrounding tissue to determine the microscopic extent of tumor spread.

TNM staging system :

This system can be applied to all primary bone tumours except multiple myeloma and juxtacortical chondrosarcoma and parosteal osteosarcoma.

T_x Primary tumor cannot be assessed. T_0 No evidence of primary tumor. T_1 Tumor confined within the cortex. T_2 Tumor invades beyond the cortex.	N_x Regional lymph node cannot be assessed. N_0 No regional lymph node metastasis. N_1 Regional lymph node metastasis.	M_0 No distant spread. M_1 Evidence of distant metastasis.

Benign bone tumors

Compact osteoma (Ivory Exostosis)

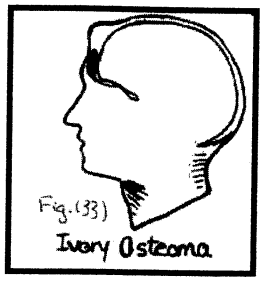
This is a rare benign tumor which arises from bones that develop from membrane. e.g. the skull.

Clinical features :

An adolescent or young adult presents with a painless, hard lump, usually on the outer surface of the skull. The tumor is rounded or conical and sessile.

Compression on neighbouring structures depends on the position of the osteoma :

1. Growth into air sinuses predisposes to infection and obstruction.
2. Deafness occurs if osteoma extends into the auditory meatus.
3. It may grow into the orbit producing distortion of the eyeball.
4. If it occurs on the inner table of the skull it may cause focal epilepsy.
5. Stretch of the overlying skin may result in alopecia or adventitious bursa formation.



Investigation :

X-ray reveals a sessile plaque of dense bone with a well-circumscribed edge.

Treatment :

- The tumor never turns malignant.
- **Indications of removal :**

1. Pressure on important structure.
2. If the patient asked for its removal.

- In the skull, an ivory osteoma should never be chiselled off, as this would result in brain concussion.

Osteoid Osteoma

- This is a painful benign osteoblastic lesion characterized by its **small size** (usually less than one cm).
- It is rounded or oval in shape.
- Patients are usually under 30 years of age and males predominate.
- Any bone except the skull may be affected, but over half the cases occur in the femur or tibia.

Clinical features : The leading symptom is **pain,** which is sometimes severe.

Investigation :

1. X-ray : Small radiolucent area, the so-called nidus, which is surrounded by dense sclerosis and cortical thickening.

2. Bone scan shows markedly increased activity.

Treatment :

The only effective treatment is complete removal of the nidus.

Osteoblastoma (Giant Osteoid Osteoma)

This is a benign lesion which differs from osteoid osteoma by :

1. It is larger in size (usually more than 1 cm).
2. It is more cellular.
3. It is usually less painful.
4. It tends to occur in the spine and flat bones.
5. **X-ray** shows a well demarcated osteolytic lesion. The surrounding sclerosis is not always easy to see.

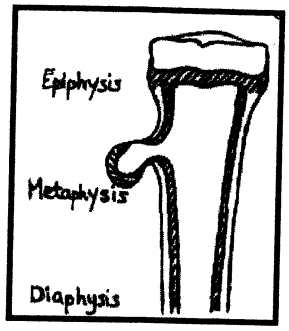
Treatment : Consists of excision and bone grafting.

Osteochondroma (Cartilage-Capped Exostosis)

- This is one of the commonest tumors of bone.

Pathology :

Origin : Chondroblast but recently it is not considered as a neoplasm but considered as a developmental anomaly (skeletal hamartoma).



Site : Metaphysis of long bones especially around the knee.

Gross picture : Smooth, pedunculated swelling of spongy bone covered by a cap of cartilage but at the age of epiphyseal fusion, it ossifies and stops to grow.

- It grows oblique away from the epiphyseal cartilage.

Clinical features :

- The usual site is the metaphysis of a long bone.
- Lesions may be single or multiple (hereditary multiple exostoses). Occasionally there is pain due to overlying bursa or parasthesia due to stretching of an adjacent nerve.

Features suggestive of malignancy :

Pain, rapid growth, invasion of the mother bone, and recurrence after excision.

X-ray :

There is a well-defined exostosis emerging from the metaphysis, its base is continuous with the parent bone. It looks smaller than it feels because the cartilage cap is usually invisible.

Treatment : If the tumor causes symptoms, it should be excised at its base.

Chondroma

- Chondroma is a benign tumour characterized by the formation of mature cartilage.
- It is usually asymptomatic and is discovered accidentally on X-ray.
- Bones affected : Most commonly short long bones of the hands and feet.
- Lesions may be solitary or multiple **(Ollier's disease).**
- It is usually situated centrally **(enchondroma)** or as an eccentric growth **(ecchondroma).**

Clinically :

The patient presents with **pain** or **a swelling** or with **pathological fracture.**

X-ray :

Shows a well-defined area in the medulla, often with characteristic specks of calcifications.

Treatment :

Is not always necessary unless it is large, presents as a pathological fracture or malignancy is suspected. The lesion should be excised or curetted and replaced with bone graft.

Giant Cell tumor (Osteoclastoma)

It occurs only in mature bone. Most commonly in the distal femur, proximal tibia, proximal humerus and distal radius, but other bones may be affected.

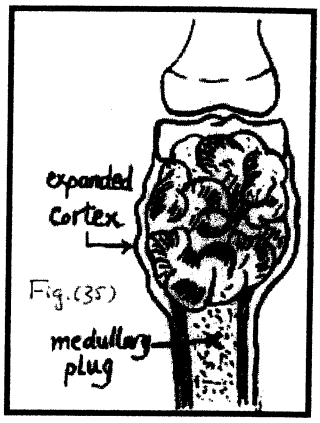


Fig.(35)

Pathology :

- The tumour has a reddish, fleshy appearance.
- It comes in pieces quite easily when curetted, but it is difficult to be removed completely from the surrounding bone.

Histologically :
Multinucleated giant cells scattered on a background of stromal spindle shaped cells which may be responsible for determining the aggressiveness of the tumor.

- **The grading system (I. II and III)** has now largely been discarded as a useful indicator of biological behaviour. Accordingly, all giant cell tumours are considered potentially malignant.

Clinical features :

Symptoms :
- The patient is usually a young adult (20-40 years of age).
- Pain at the end of a long bone.
- Sometimes there is slight swelling.
- There may be a history of trauma and pathological fracture is reported in 10 - 15% of cases.

Signs :
- There is swelling at the end of a long bone.
- The consistency depends on the degree of thinning of the expanded cortex, it may be soft, firm or an egg-shell crackling sensation may be detected.
- The neighbouring joint is often irritated.

Investigation :

1. X-ray : A radiolucent area situated eccentrically at the end of long bone. It has the characteristic **soap bubble appearance.** Important is the presence of the **medullary plug (operculum)** at the junction of the shaft with the tumour. The lack of this plug may signify malignant osteoclastoma.

2. Detailed staging procedures are essential. **CT scans** and **MRI** will reveal the extent of the tumor, both within the bone and beyond.

3. Arthroscopy & biopsy may be helpful to establish affection of articular surface.

Treatment :

1. The simplest treatment is **curettage and bone grafting,** but recurrence is common.
2. Wide excision is the treatment of choice, with replacement by specially designed prosthesis or by osteocartilaginous grafts.
3. Amputation is indicated for tumours which recur with increasing evidence of malignancy.
4. Radiotherapy is reserved for surgically inaccessible tumors.

Osteosarcoma

- Osteosarcoma is a highly malignant tumor characterized by the formation of bone osteoid tissue.
- It is the most common primary tumor of bone.
- **Bone affected :** It may affect any bone but most commonly involves the long bone metaphyses, especially around the knee and at the proximal end of the humerus.

Pathology :

- The tumor is usually situated in the metaphysis of a long bone, where it destroys & replaces normal bone.
- **The rule of 80 :** 80% of osteosarcoma occur in teenagers, 80% in the lower limb, 80% around the knee, 80% in the lower end of the femur, 80% in the metaphysis.

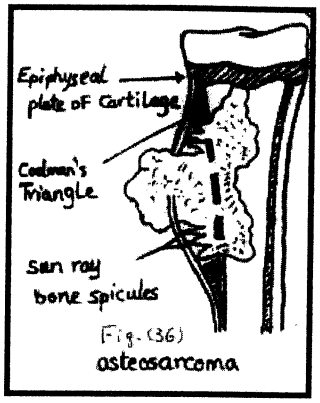


Fig. (36) osteosarcoma

- **The cell of origin** is the primitive osteoblasts.
- The tumor rapidly spreads towards the shaft, but it respects the epiphyseal cartilage and hence does not invade epiphysis or the joint.

- **There are four main pathological features :**

1. Bone destruction.
2. Tumour bone formation (radiologically appears as **sun-ray appearance**).
3. Reactive bone formation (radiologically appears as **Codman's triangle**).
4. Soft tissue infiltration.

Predisposing factors :

1. Irradiation.
2. Paget's disease of bone.

Types :

1. Osteolytic type : Soft, fleshy and vascular with areas of hemorrhage and necrosis.
2. Sclerosing type : Solid and contains bone.

Histological appearances :

- It shows considerable variation :

Some areas may have the characteristic spindle cells with a pink-staining osteoid matrix; others may contain cartilage cells or fibroblastic tissue with little or no osteoid.

Osteosarcoma variants :

1. Parosteal (juxtacortical) osteosarcoma :

- Characterized by an origin on the external surface of bone. The tumor commonly arises around the knee with a slow growing rate to form a very large mass which encircles the bone.

2. Periosteal osteosarcoma :

- Is a rare type that arises as a surface tumor, most commonly in the tibial shaft.
- There is involvement of a short segment of cortex and the medullary cavity is not involved.
- Prognosis : Intermediate between that of parosteal and conventional osteosarcoma.

3. Osteosarcoma in Paget's disease :

- This is usually a high grade anaplastic osteosarcoma.
- It mostly involves the pelvis, humerus, femur and vertebra.

4. Post irradiation sarcoma of bone.
5. Intraosseous low grade osteosarcoma.
6. Intraosseous high grade osteosarcoma.

7. Small cell osteosarcomas :
- Is a recently described variant which shows the histological features of both Ewing's sarcoma and osteosarcoma. The tumor behaves more like osteosarcoma and is not radiosensitive.

Clinical features :

- **The incidence** of osteosarcoma is highest between the age of 10 and 20 years, but a second peak occurs after 50 years of age due to malignant changes in Paget's disease.
- **Pain** is usually the first symptom; it is constant, worst at night & gradually increases in severity.
- Sometimes, the patient presents with **a lump** but **pathological fracture is rare** because the patient is bed ridden due to the pain
- **On examination :** Local tenderness, In later cases there is a palpable mass and the overlying tissues may appear swollen and inflamed.
- **The regional lymph nodes may be enlarged.**

Investigations :

1. Laboratory : The ESR is usually raised & there may be increase in serum alkaline phosphatase level.
2. X-ray may show :
- Streaks of new bone formation, radiating outwards from the cortex **(sun-ray appearance).**
- Reactive new bone formation at the angles of periosteal elevation **(Codman's triangle).**

Both sunray appearance and Codman's triangle are typical of osteosarcoma, yet they may occasionally be seen in other rapidly growing tumours.
- Hazy osteolytic areas affecting the cortex **(ghost of cortex).**

3. CT and MRI reliably show the extent of the tumour.
4. Chest X-ray may show pulmonary metastasis.
5. Biopsy is essential to establish the diagnosis. (see before)

Differential diagnosis :

1. Chronic pyogenic osteomyelitis.
2. Giant cell tumour.
3. Other malignant bone tumors as chondrosarcoma, fibrosarcoma, Ewing's sarcoma and reticulum cell sarcoma.
4. Secondary carcinomatous deposits.

Treatment :

1. Local control of the disease is by either amputation or wide local excision and prosthetic replacement.
- The level of amputation should be proximal to the joint above the tumour, e.g. osteosarcoma at the tibia is treated by an above knee amputation.

2. Adjuvant chemotherapy has improved the prognosis.

Chondrosarcoma

- Chondrosarcoma is a malignant tumor characterized by the formation of cartilage.
- It can occur either as a primary tumour **(primary chondrosarcoma)** or as secondary change in a pre-existing cartilaginous lesion: multiple enchondromatosis **(secondary chondrosarcoma).**
- **Age :** Both types have their highest incidence in the fourth and fifth decades and men are affected more often than women.
- The entire tumor is slowly growing and patients may complain of a dull ache or a gradually enlarging lump. Medullary lesions may present as pathological fracture.

X-rays :

1ry Type : Destructive medullary tumour containing characteristic flecks of calcification.
2ry Type : Shows the bony exostosis often surrounded by clouds of patchy calcifications in the otherwise unseen lobulated cartilage cap. Calcifications are very fluffy & poorly outlined.

Treatment :

1. Wide local excision.
2. Where neither excision nor amputation is feasible, radiotherapy may be used **(chondrosarcoma has a very poor or no response to chemotherapy or radiotherapy).**

Fibrosarcoma of Bone

- Fibrosarcoma is rare in bone and is more likely to arise in previously abnormal tissue (bone infarct, fibrous dysplasia or after irradiation).
- It occurs in older patients than do osteosarcoma (20 - 60 years of age).
- The patients complains of pain, swelling and there may be a pathological fracture.
- X-ray shows an area of bone destruction.

Treatment :

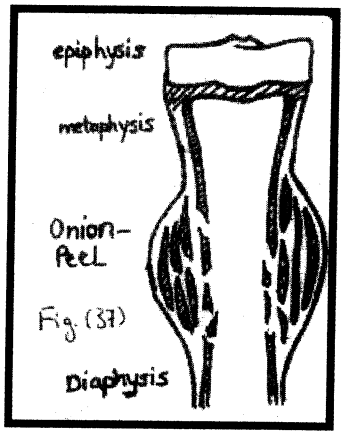
1. Wide excision with local prosthetic replacement (in law grade, well-confined tumors).
2. Radical excision or amputation (in high grade lesions).
3. Radiotherapy.

Ewing's Sarcoma

- This is a rare malignant tumor arising from vascular endothelium in the bone marrow.
- It usually occurs in the diaphysis of a long bone and gives rise to marked periosteal reaction.

Pathology :

Gross picture :
The tumor arises in the medulla as a semisolid greyish white mass which spread longitudinally and transversely raising the periosteum with subperiosteal new bone formation in successive layers parallel to the shaft **(onion peel appearance in X-ray).**



Cut section : Ill-defined greyish white mass with areas of hemorrhage and necrosis.

Microscopic picture : Small rounded cells arranged in rosette around the blood vessels.

Clinical features :

- **Age :** Between 10-20 years usually in a tubular bone, and especially in the tibia, fibula or clavicle.
- **Pain and swelling** are the chief presenting features.
- **The lump is warm & tender, ill-defined & is diaphyseal with intermittent or continuous pyrexia, elevated ESR & high leucocytosis.** ***The clinical features may resemble those of osteomyelitis.***
- **Blood born metastasis** may occur to other bones and in lungs.
- **Lymphatic spread** to regional lymph nodes is common.

Investigations :

1. Elevated ESR & Leucocytosis.

2. X-ray usually shows an area of bone destruction which is diaphyseal with new bone formation in layers along the shaft **(onion peel effect).**

- **More often the tumour extends into surrounding soft tissues and may show sunray appearance and Codman's triangle.**

3. Bone scan may show multiple areas of activity in the skeleton.

4. CT and MRI reveal the local extent of the tumor.

Differential diagnosis :

1. Secondaries from neuroblastoma. Usually the patient is below 5 years of age.
2. Reticulum cell sarcoma. Usually occurs in patients above 20 years of age. There are reticulin fibers and no intracellular glycogen in tumour cells.
3. Osteomyelitis : Biopsy will establish the diagnosis.

Treatment :

The prognosis is always poor. The best results are achieved by combination of chemotherapy, surgery and radiotherapy; then a further course of chemotherapy for one year is given.

Metastatic Bone Disease

- Metastatic deposits are more common than all primary bone tumours.
- **Sources :** The commonest is carcinoma of the breast, prostate, kidney, lung, thyroid, bladder and gastrointestinal tract.
- In 10% of cases no primary tumour is found.
- Metastases usually appear in areas containing red marrow.
- It is usually osteolytic, may be osteoblastic (carcinoma of the prostate & some breast carcinoma).

Clinical features :

- **Age :** Patients are usually aged 50-70 years.
- **Clinical picture :** Pain (the commonest and often the only clinical feature), pathological fracture.
- The primary tumor may be obvious, but sometimes even a meticulous search fails to reveal it.
- **Symptoms of hypercalcemia may occur :** Anorexia, nausea, abdominal pain, general weakness, depression and polyuria.

Investigations :

1. Serum alkaline phosphatase is often increased, and **in prostatic carcinoma the acid phosphatase is elevated.**

2. X-ray. Most skeletal deposits are osteolytic .

3. Bone scan is the most sensitive method of detecting silent metastatic deposits in bone.

Treatment :

Treatment is palliative including chemotherapy, radiotherapy and :

1. Control of pain and metastatic activity by radiotherapy.
2. Pathological fractures are treated by combination of internal fixation & radiotherapy.

Fractures & dislocations of the spine

They are important as they may produce spinal cord and nerve root injuries. Fractures are more frequent at the junction of the mobile and rigid parts in the lower cervical and dorso-lumbar regions.

Etiology :

[A] Trauma :

1. Hyper-flexion trauma : This is the most frequent type of trauma
2. Hyperflexion and rotation.
3. Vertical compression trauma.
4. Hyper-extension injuries are uncommon.

[B] Pathological fractures : Metastases & Osteoporosis.

Types :

I- According to morphology :

1. Wedge compression fracture.
2. Fracture dislocation.
3. Dislocation.
4. Comminuted (burst) fracture.
5. Avulsion fractures of transverse and spinous processes of vertebrae.

Dislocations and fracture dislocations of the atlas and axis vertebrae are especially serious as they may be fatal because of transection of the cord above the level of innervation of the respiratory muscles.

II- According to stability :

1. Stable fractures have intact posterior ligaments e.g. wedge compression fracture.
2. Unstable fractures have torn posterior ligaments and are liable to injure the spinal cord and nerve roots e.g. fracture dislocations and pure dislocations.

Neurological injury :

Paraplegia or quadriplegia. It is important to diagnose the level of the lesion as well as the nature to know whether recovery is expected or not.

Level of lesion : (see internal medicine).

Nature of the lesion :

1. Spinal cord concussion (like neurapraxia) : recovery is the rule.

2. Spinal cord damage by contusion or laceration (transection) : does not recover. (clinical picture see internal medicine).

The types of spinal cord transaction are :

(a) Complete transaction.
(b) Hemisection (Brown-Séquard syndrome).

3. Cauda equine injury.

Clinical features :

1. Spine injury : is suspected after any major trauma or if the victim is unconscious after the injury. A cervical collar is applied before moving the victim and the head is immobilized.

2. Other injuries : of the skull, chest, abdomen, or limbs must be searched for.

3. Local signs : The patient is gently rolled onto his side as one piece.
(a) A slight kyphosis may be visible or felt.
(b) There is local-tenderness over the spinous process. No gap is palpable between the spinous processes in a stable fracture. In an unstable fracture, separation of the spinous processes may be evident.
(c) No attempt should be made to test back movements as this may induce paraplegia.

4. Neurological assessment :
(a) Motor power.
(b) Sensations.
(c) Reflexes.

Radiography :

1. Plain X-ray
2. CT scan and MRI : must be done to assess encroachment on the spinal cord.

Treatment :

[A] Initial management :

1. When fracture of the spine is suspected, movements of the spine must be prevented.
1. A cervical collar is applied and the head is immobilized during transport and during examination.
2. The trauma victim is moved on to the stretcher all in one piece.

2. Shock must be treated.
3. Associated injuries may require urgent treatment.

[B] Stable fractures :

1. Wedge compression fracture : No interference concerning the fracture. The patient is put to bed and is taught extension exercises for the back muscles. Within a month, he is allowed to move. He can resume work in 3 months. A painless, mobile and powerful back is achieved in spite of the fact that the compressed vertebra remains wedge shaped.

2. Comminuted fracture : Pain is usually severe and healing is delayed. A plaster Jacket is applied in the neutral position (straight spine; not hyperextended) for 3 months. The patient can walk with the plaster on.

3. Avulsion fractures of the transverse or spinous processes : Rest and Analgesics.

[C] Unstable fractures :

No neurological deficit

1. Cervical spine : Traction for 6 weeks is followed by a cervical collar for one month. Open reduction and internal fixation by plates may be needed.
2. Thoraco-dorsal spine : Reduction by gentle extension is followed by fixation in a plaster cast in mild extension. If closed reduction is not possible, open reduction and internal fixation is the alternative.

[D] Evident cord transaction : (see internal medicine for paraplegia treatment).

Potts' Disease Of The Spine

Incidence :

- The spine and the hip are the commonest two joints to be affected by tuberculosis.
- The disease is commonest in children (75% below 10 years) but may occur at any age.
- In adults the prognosis is bad.

Pathology :

Site :

- The commonest site is the **lower thoracic** and **upper lumbar** being :
  1. The junction of fixed and mobile parts.
  2. The close proximity of the thoracic duct and cisterna chyli which drain the tubercle bacilli from the abdomen.
- Usually **two or three vertebrae** are affected with the discs in between.
- **In children,** the disease starts in the cancellous bone in the centre of the vertebral body.
- **While in adults,** the disease starts subperiosteal under the anterior longitudinal ligament.

Complications :

- **Because the destruction is more in the anterior part of the vertebra and disc, away from the spinal cord, the disease leads more to deformity rather than to pressure on the spinal cord and paraplegia.**

1. Deformity (common) : Kyphosis most marked in the thoracic region **(gibbus).**

2. Cold abscess : Under the anterior longitudinal ligament (paravertebral cold abscess). It travels in the lines of least resistance guided by the fascial planes :

(a) In the cervical region : Retropharyngeal abscess.

(b) In the thoracic region : Accumulates in the mediastinum, present on the side of the chest wall or on the sides of the sternum.

(c) In the lower thoracic and the lumbar regions : It passes into the sheath of the psoas muscle to form iliopsoas abscess which presents as a cystic swelling in the posterior abdominal wall, or it may pass under the inguinal ligament to present in the femoral triangle lateral to the femoral vessels with cross fluctuation between the two collections. It may be mistaken for a femoral hernia.

3. Paraplegia : Occurs in 10% of cases. It is commonest in the upper dorsal spine, being the narrowest part of the spinal canal.

Paraplegia complicating Pott's disease is classified into :

Items	Early-onset paraplegia	Late-onset paraplegia
Due to :	1. Edema, thickening & congestion of the dura. 2. Pressure by an abscess. 3. Bony sequestrum.	1. Increasing deformity. 2. Reactivation of disease. 3. Vascular insufficiency of the cord.
Treatment :	Early anterior decompression and debridement followed by spinal fusion. About 80% recover within few weeks.	If the MRI shows a block, operative debridement is done. If there is no block, operation is unlikely to be of use.

Clinical picture :

1. Marked tuberculous toxaemia : wasting and fever.
2. Local pain : which increases on movement.
3. Limitation of movements in all directions (the most important signs) :
(a) A child is unable to pick a coin form the ground with the knees straight.
(b) When the child is laid on his face and the ankles grasped and raised, the spine is raised as one piece without the lumbar curve.
4. Deformity :
- Most marked in the thoracic region, it is an angular deformity (gibbus).
- In the cervical and lumbar regions the deformity is masked by the normal lordosis.

5. Cold abscess.

Investigations :

I- Laboratory : 1. CBC. 2. ESR. 3. Tuberclin test.
II- Chest x-ray.
III- X-ray of the spine :
1. Two or more vertebrae are affected.
2. Intervertebral disc space is lost.
3. Adjacent surfaces of the vertebrae are irregular.
4. Vertebral bodies may be collapsed with areas of bone destruction.
5. No new bone formation.
6. Paravertebral abscess shadow is usually preset.

IV- CT scan & MRI.

Differential diagnosis :

1. Rickety kyphosis.
2. A neglected fracture spine.
3. Secondaries in the spine.
4. Lumbar Potts has to be differentiated from hip disease. Both produce flexion of the thigh and psoas spasm. In hip disease, hip movements are limited in all directions.

Treatment :

[A] Conservative treatment :

1. Anti-tuberculous drugs for 9 months.
2. Rest & good nutrition.
3. Spine support by blaster cast for a short period.
4. Periodic clinical, hematologic & radiologic assessment.

[B] Surgery :

Indications :

1. Early paraplegia with No sign of recovery after 3-4 weeks of conservative treatment.
2. Paraplegia getting worse in spite of conservative treatment

Aim of surgery : Decompression of the spinal Cord.
Technique :
- The caseous material and dead bone are removed by one of 2 methods :

1. Costo-transversectomy : in this operation the posterior ends of one or two ribs and the corresponding transverse processes of the vertebrae are excised and the cold abscess is evacuated.
2. Antero-lateral decompression : in this operation, in addition to costotransversectomy the pedicles & part of the vertebral bodies are excised to achieve decompression.

[C] Later treatment :

1. Life long follow-up.
2. Spine fusion : may be needed after clearance of infection, using bone graft.

Neurosurgery

Neurosurgery

Head Injuries

Classification :

[A] Scalp Injuries : Hematoma and Wounds.
[B] Skull : Fractures which may be :
1. Simple. 2. Comminuted. 3. Depressed. 4. Compound. 5. Complicated.
[C] Dura : Laceration.
[D] Brain :
1. Concussion. 2. Contusion. 3. Laceration. 4. Compression. 5. Edema.
[E] Blood Vessels : Hemorrhage from arteries , veins or sinuses.
1. Extradural. 2. Subdural. 3. Subarachnoid. 4. Intracerebral. 5. Intraventricular.
[F] Cerebrospinal Fluid :
1. Lost through the nose (rhinorrhea) or the ear (otorrhea).
2. Blood stained due to subarachnoid hemorrhage.
3. Infected (Meningitis).
4. Obstructed in circulation (Hydrocephalus).
[G] Cranial Nerves : 1. Traction injury. 2. Compression.

Scalp Injuries

Wounds of the scalp :

These may be contusions , hematomas , lacerations or incised wounds.
- Bleedeing is very excessive to the extent of causing shock.
- Healing is rapid.
- If infection occurs in the area of the loose areolar tissue , extensive cellulitis is likely to occur.

Management :

[A] How to stop bleeding from a scalp wound ?
1. **Direct pressure.** A depressed fracture should be excluded before applying direct pressure.
2. **Applying multiple Allis forceps to the edge of the wound** and bending them.
3. **Applying multiple artery forceps to the galea aponeurotica** and allowing them to fall back over the skin edge.

- All these measures are temporary before suture of the wound.

[B] If there is a possibility of a skull fracture , plain skull X-Ray is performed.
[C] Closure is in 2 layers :
1. Galea to galea by absorbable sutures.
2. Skin to skin by non-absorbable sutures.

Hematoma of the scalp : 3 types :

Subcutaneous Hematoma	Subgaleal Hematoma	Subpericranial Hematoma (Cephalhematoma)
Confined to the dense subcutaneous layer.	Occupies the loose areolar tissue under the epicranial aponeurosis.	Under the periosteum.
Small , Painful. Moves with the scalp over the skull.	Large , Soft , Fluctuating swelling reaching Anteriorly to the supraorbital ridges , Posteriorly to the nuchal lines & Laterally to the temporal line. The scalp floats over the swelling.	Limited by the suture line to the underlying bone usually the parietal.

Treatment :
- **Subgaleal Subpericranial Hematoma :** Aspiration and a pressure bandage.
- **Subcutaneous Hematoma :** Will resolve spontaneously.

Fractures of the skull vault

Causes :

- **Always direct trauma.** There are 3 mechanisms :

1. Local indentation : Trauma by localized objects (e.g. hammer) produces depressed fracture.
2. Compression of the skull (e.g. car accident) : Bursts skull producing fissure fractures or separated sutures.
3. Missile injuries.

Pathology :

1. Fracture : may be :
(a) Closed (Simple) : No scalp wound.
(b) Open (Compound) : With scalp wound.
2. Fracture : may be Fissure or Depressed (rarely Avulsion).
3. Open fractures (fissure or depressed) carry the risk of :
(a) Escape of intracranial contents (Blood , CSF , Brain matter).
(b) Introducing foreign bodies to brain.
(c) Introducing infection : Meningitis , Encephalitis.
4. Associated Brain Injury : Concussion , Contusion , Laceration , Compression.

Clinical Picture : 3 Groups of signs in each type :

Closed (Simple) Fissure Fracture	Closed (Simple) Depressed Fracture
1. Hematoma over it.	1. Hematoma over it.
2. Fissure can't be felt through intact scalp.	2. depression may be felt through intact scalp. (DD Subpericranial Hematoma).
3. Sings of Associated Brain Injury.	3. Sings of Associated Brain Injury.
Open (Compound) Fissure Fracture	**Open (Compound) Depressed Fracture**
1. Escape of Blood , CSF or Brain matter.	1. Escape of Blood , CSF or Brain matter.
2. Fissure can be seen and felt by gloved finger. It differs from suture line which is : (a) In a special site. (b) Zigzag shape. (c) Not bleeding.	2. Depression can be seen and felt.
3. Sings of Associated Brain Injury.	3. Sings of Associated Brain Injury.

- **Concussion is surprisingly slight and there is usually no compression. The main hazard here is the ability to infection.**

Investigations :

- **X-Ray :** Shows Fracture type , site , size & shape.

1- 1. Fissure differs from suture line in site and shape : suture has a serrated end and exists at certain sites.
2. Indepressed fractures the following are determined :
(a) Site is important : over speech or motor area.
(b) Size : Large depressed fracture may cause compression.
(c) Degree of depression :
i. Pond fracture with no sharp edge.
ii. Gutter with sharp depressed edge produces brain laceration.

Complications of a depressed fracture :

1. Dural tear : leading to prolapse of the brain.
2. Infection : this is very serious as it may lead to osteomyelitis or meningitis.
3. Epilepsy : either early or late.

- Elevation of the depressed segment may diminish but doesn't abolish this complication.

Items	Early Epilepsy	Late Epilepsy
Time	Within 1 week after the injury.	Within 1-4 years after the injury.
Incidence	Increased in patients with depressed fracture or intracranial hematoma.	Is much higher in those who had early epilepsy.
Ttt	These patients need prophylactic anticonvulsants for 6 weeks.	Long term anticonvulsants.

4. Cosmetic deformity.
5. Severe bleeding : from one of the venous sinuses.
6. Associated Brain Injury.

Treatment : (Mainly directed to brain injury)

[1] Simple Fissure Fracture :

1. Observation for evidence of intracranial injury is done.
2. The site of a fissure fracture (e.g. overlying the course of the middle meningeal vessels) should alert the surgeon to the possibility of an extradural hematoma.

[2] Simple Depressed Fracture :

- Operative elevation of the depressed fragment is indicated only in the following conditions :

1. If depression is **large.**
2. Sharp inner edge seen by X-Ray.
3. If the fracture is overlying **an air sinus.**
4. If the fracture is overlying **an important area** e.g. Speech or Motor area.
5. If there is a possibility of a **dural tear.**
6. If it is associated with **cerebral compression.**
7. If the depressed segment is causing a **cosmetic deformity** e.g. in the frontal bone.

[3] Compound Fissure Fracture :

- Treated by **Excision** of the scalp wound and its **Suture** followed by **Observation** of the patient for evidence of associated brain damage.

[4] Compound Depressed Fracture :

- These patients are operated upon in the theatre under absolute aseptic precautions :

1. Foreign bodies are meticulously removed.
2. The depressed segment is **Gently** elevated to avoid tearing of the dura.
3. Any prolapsed or necrotic brain tissue is sucked and hemostasis is performed.
4. Any dural tear is repaired.
5. Removed bone segments are cleansed and replaced.
6. The pericranium and the scalp are sutured.
7. Prophylactic antibiotics are administered.

NB : The most important cover to the brain is the scalp and every attempt should be made to preserve and close it.

Fractures of the skull base

Causes :

1. Usually indirect trauma : Trauma to the vault breaks base by 2 mechanisms :

(a) Bursting : As skull is compressed , vault escapes injury being elastic. Base breaks being rigid and weakened by foramina.

(b) Radiation : Trauma produces fissure in the vault which spreads to base.

- These fractures are usually secondary to a major trauma and are usually accompanied by a seriuos brain injury.

2. Less commonly direct trauma : Penetrating sharp objects or bullets through orbit , nose or mouth.

Pathology :

1. The fracture is usually compound (i.e. opening to the exterior through nose or ear) causing :

(a) Escape of intracranial contents (Blood , CSF , Brain matter).

(b) Risk of meningitis.

2. Cranial nerve injury.

3. Brain injury : Concussion , Contusion , Laceration or compression.

Clinical Picture :

1. Clinical Picture of Associated Brain Injury.

2. Hemorrhage :

(I) In Anterior Cranial Fossa , Bleeding comes from :

(a) Bleeding from nose (epistaxis).

(b) Bleeding from mouth.

(c) Orbital bleeding : with ecchymosis of lid & subconjunctival hemorrhage **(Panda Bear or Raccoon sign). DD Traumatic Black Eye.**

- **Subconjunctival Hematoma differs from Black Eye of direct trauma in :**

1. No skin bruises around orbit.
2. It occurs several hours after the accident.
3. It starts in lower eyelid.
4. Subconjunctival hematoma is triangular with apex at lid margin and base extending deeply beyond lid base.
5. In traumatic black eye :
   (a) Proptosis may occur due to retro-ocular hematoma. (b) Limited eye movements.

(II) In Middle Cranial Fossa , Bleeding comes from :

(a) The ear : associated with CSF otorrhea.

(b) The nose (epistaxis) : fracture involves the sphenoid bone.

(III) In Posterior Cranial Fossa : Blood accumulates subcutaneously posterior to the mastoid process.

3. Escape of CSF from the nose (rhinorrhea) or ear :

- It is due to tear in the dura.
- The rhinorrhea may persist for several weeks, and air may enter the cranial cavity when the patient blows his nose **(Intracranial Aerocele).**
- CSF fluid is salty , alkaline , its Sp. Gr. is 1005 , it doesn't coagulate on boiling and contains glucose.

4. Escape of brain matter from the nose or mouth or ear.

5. Cranial Nerve Injury : **ALL** cranial nerves are liable to be injured except the 12th because the condyloid foramen is protected by ridge of bone which deviates line of fracture into the foramen magnum.

(a) In Anterior Cranial Fossa : The first 6 cranial nerves may be injured.

(b) In Middle Cranial Fossa : The Facial & Auditory nerves may be injured.

(c) In Posterior Cranial Fossa : The Vagus & Glossopharyngeal nerves may be injured.

Treatment : **3 Main Lines :** (X-Ray doesn't show the fracture)

1. Prevention of infection : Antibiotics. Bleeding ear is plugged by sterile cotton.

2. Control of CSF leakage by :

(a) Semi-sitting position.

(b) Warn the patient not to blow the nose.

(c) If leakage from nose persists after 10 days, torn dura is repaired or grafted by fascia late to avoid meningitis.

3. Management of Associated Brain Injury.

Brain Injuries

Causes :

1. Open injuries : Sharp objects or bullets produce cerebral laceration or hemorrhage.

2. Closed injuries.

Surgical Pathology :

Mechanism of injury :

The main factor which leads to most of the sequelae of head injury is the dispalcement and distortion of the cerebral tissues. **The severity of cerebral injury is determined by :**

1. Distortion of the brain.

2. Mobility of the brain in relation to the skull and membranes.

3. The configuration of the interior of the skull : Smooth areas of the skull will cause less damage.

4. Age of the patient : A young patient will have a better chance of recovery.

Pathology :

[A] Primary Pathological Sequelae :

Brain Concussion , Contusion & Laceration represent different degrees of brain damage which occurs at the moment of impact.

[B] Secondary Pathological Sequelae :

1. Brain edema : Clinically this simulates the picture of brain compression without focal manifestations.

2. Intracranial hemorrhage : This may be extradural , subdural or intracerebral.

3. Infection.

4. CSF rhinorrhea.

5. Coning : The rise of ICT will cause herniation of the contents of the supratentorial compartment through the tentorial hiatus , or contents of the infratentorial compartment through the foramen magnum.

6. Brain stem injury : The patient is unconscious with spontaneous extension spasms of all 4 limbs , tachycardia , small pupils , pyrexia and rapid shallow breathing. With intensive treatment , the patient may recover but there is often marked spasticity.

7. Vascular changes : With resultant ischemic necrosis and subsequent brain edema.

Extracranial factors that affect the cerebral injury

1. Respiration : hypoxia and increased PCO2 can aggravate the development of severe brain edema and venous congestion.

2. Blood volume & blood pressure : hypovolemia may lead to irreversible cerebral damage.

3. Fluids : intravenous infusion of hypotonic fluids leads to increased brain edema.

4. Temperature : rise in body temperature will cause deterioration in neurological state due to increased metabolic rate.

Cerebral Concussion

• **Transient loss of consciousness with paralysis of all cerebral functions which follows immediately after injury.**

• It is due to diffuse neuronal injury from excessive stretching of cerebral tissues during sudden displacement of the brain (affecting the activation portion of the reticular formation).

Clinical Picture :

1. Sudden loss of consciousness with relaxed muscles and closed eyes.
2. Skin : pale , cold & clammy.
3. Respiration : slow , shallow.
4. Pulse : rapid , weak.
5. Temperature : subnormal.
6. Reflexes are lost & Sphincters relaxed.
7. Recovery occurs gradually and is usually complete and perfect.

Cerebral Contusion

• Multiple surface contusions in the frontal and temporal lobes with rupture and degeneration of the axons with focal damage of cortical or hypothalamic levels of the brain.

Clinical Picture : Is that of Concussion but :

1. Unconsciousness is prolonged.
2. Recovery of consciousness is incomplete (confusion or irritability).
3. Local signs of brain damage occur e.g. monoplegia , aphasia.
4. In severe cases , deep coma persists till death.

Cerebral Laceration

Causes :

1. Blows on the head causing fracture. Injury is either beneath the point of trauma or on the opposite side (counter-coup).
2. Penetrating wounds.

Clinical Picture :

1. Symptoms of concussion merging into compression or followed by irritation.
2. Convulsion affects regular groups of muscles and is followed by permanent paralysis.

Acute Extradural Hematoma

Etiology :

- Usually this lesion is due to **a trivial trauma applied to the side of the skull** causing a fracture of the temporal or parietal bone.
- The dura is driven inwards and tearing of the anterior or posterior branches of **the middle meningeal artery or vein** occurs leading to accumulation of blood in the extradural space.

Blood will pass in 3 directions :

1. Outwards : through the fracture to form a boggy swelling under the temporalis muscle.
2. Upwards : over the parietal region.
3. Downwards : into the middle fossa.

- **An extradural hematoma can occur without a skull fracture especially in children as they have an elastic skull.**

Clinical Picture :

The Clinical Picture is usually described as passing into 3 stages :

[I] Stage of Concussion : The patient usually has a mild blow to the head (e.g. a ball hitting the skull) causing a brief period of concussion.

[II] Stage of Lucid Interval : The patient recovers from the initial concussion and may even resume his activities. During this period , blood is accumulating gradually in the extradural space.

[III] Stage of Compression :

1. A rising ICT produces the following manifestaions :
(a) Deterioration in the level of consciousness. (b) Slowing pulse rate.
(c) Rising BP. (d) Slowing respiration.

2. Gradual progressive deterioration in the level of consciousness is the main feature. The patient becomes confused and irritable. Later he becomes drowsy, semi-comatosed or even comatosed with stertorous breathing.
3. Contralateral hemiparesis : due to compression of the ipsilateral cerebral cortex.
4. Tentorial herniation occurs. Compression of the oculomotor nerve causes constriction, rapidly followed by dilatation of the ipsilateral pupil. Later the contralateral pupil will constrict and then dilate.
5. Continuous tentorial herniation forces the opposite crus against the rim of the tentorium, producing hemiparesis, which this time occurs on the same side of the hematoma.
6. As the coma deepens, the BP rises and the pulse & respiration slows down.
7. Finally, hyperpyrexia, decebrate rigidity and bilateral dilated fixed pupils occur, and constitute clinical evidence of bad prognosis.

- It should be stressed that this classic picture is only found in a minority of patients.
- If the source of bleeding is venous, the lucid interval will be longer.

Signs of lateralisation (Signs denoting side of compression)

1. Side of hematoma beneath temporalis muscle.
2. Side of skull fracture and shift of penial body (Plain X-Ray).
3. Side of initial constriction and dilatation of the pupil.
4. Contralateral side of convulsions or paralysis.
5. Urgent investigations in difficult cases :
   (a) Carotid angiography.
   (b) Ultrasonic encephalography.
   (c) EEG.
   (d) CT scan & MRI.
   (e) Indoubful cases , trephine exploration is essentail on both sides.

Acute Subdural Hematoma

- This usually follows a severe blow to the skull leading to rupture of a large cortical vein as it crosses the subdural space to reach the fixed subdural venous sinuses.
- The hematoma is less commonly due to a cortical laceration with subsequent hemorrhage.
- Clinically the patient may present with a picture similar to that of an extradural hematoma but certain differences exist.

Differences between acute extradural & subdural hematoma:

Acute Extradural Hematoma	Acute Subdural Hematoma
Usually mild trauma.	Severe trauma.
Usually mild brain damage.	Severe brain damage & laceration.
Lucid interval may be present.	Persistent loss of consciousness , No Lucid interval.
The hematoma is usually unilateral.	Commonly bilateral & extensive.
Early surgery is successful.	The patient has serious brain damage & edema in addition to the hematoma; so the results of surgery are not very successful. Mortality rate is up to 50%.

NB : CT scan should be performed before surgery to evaluate the size of the hematoma and the degree of brain damage & edema.

Management of Cerebral Injuries

The management of patients with a head injury must follow a planned program as follows:

1. Dealing with the life saving priorities.
2. Initial examination.
3. Performance of the necessary investigations (only after resuscitation).
4. Continuing care & observations.
5. Possible need for surgery to evacuate an intracranial hematoma.

[1] Dealing with life saving priorities : {ABC}

[A] Air way:

- **Air way obstruction may result from :**

1. Inhaled blood , CSF , vomitus , mucus.
2. Falling back of tongue.
3. Retained secretions.
4. Paralyzed vocal cords (from injured vagus).

- **Clear air way is maintained by :**

1. Pulling on a fallen tongue + oral airway.
2. Regular aspiration of secretions.
3. Endotracheal tube or even urgent tracheostomy may be needed.

[B] Breathing :

- **Take care that difficult breathing causes brain congestion which leads to :**

1. Increased CSF.
2. Increased bleeding.
3. Brain edema.

[C] Circulation : Treatment of shock if present.

[2] Initial examination :

- **What to examine ?**

1. Vital signs : Pulse , BP & Respiratory Rate.
2. Chest & Abdomen for associated injuries.
3. Scalp for any scalp wound or hematoma.
4. Skull for any fracture.
5. Assessment of the head injury.
6. Pupils : for their size & reaction to light.
7. Limbs : for any fractures or vascular injury.
8. Back : for possibility of fractures & dislocations.
9. Assessment of level of consciousness **(GCS).**

GLASCOW Coma Scale (GCS) : It is based on :

Eye opening		Best verbal response		Best motor response	
Spontaneous	4	Oriented	5	Obeys command	6
To verbal command	3	Confused	4	Localizes pain	5
To pain	2	Inappropriate words	3	Flexion withdrawal	4
None	1	Incomprehensible sounds	2	Abnormal flexion	3
		None	1	Abnormal extension	2
				None	1

- The total points are added. A score less than 8 have a very guarded prognosis.

- **Indications for admission to the hospital of patients with head injury include :**

1. Any depression of level of consciousness.
2. Skull fracture.
3. Focal neurological signs.
4. Persistent headache or vomiting.
5. Absence of responsible relatives who can observe the patient for the first 24 hours.

[3] Performance of the necessary investigations (only after resuscitation) :

(A) Plain skull X-Ray. Value :

1. Can demonstrate the site and type of skull fracture.
2. May give a clue for the severity of case.
3. Help in detecting the possible site of an extradural hematoma.
4. Can visualize foreign bodies.

(B) CT scan. It can diagnose :

1. Fractures of the vault or base of skull.
2. Brain contusion or laceration.
3. Brain edema.
4. Intracranial hematomas.

If the patient, on clinical grounds, is in urgent need for evacuation of an intracranial hematoma, no time should be lost in doing investigations & surgery should be done immediately.

[4] Continuing care & observations :

1. Attention to air way (tracheostomy may be needed).
2. Nasal feeding through Ryle's tube.
3. Catheterisation if necessary.
4. Continuous observation : every half an hour for Pulse , Level of consciousness , Pupils & Power of movements on the 2 sides of the body. Progressive slowing of pulse and deterioration of consciousness indicate cerebral compression. The appearance of new localising signs (e.g. pupil changes or paralysis) indicates focal compression by hemorrhage. Immediate exploration by burr hole or trephine is indicated.
5. Drugs :

(a) Osmotic diuretics : to reduce brain edema.

- 250 ml of 20% Mannitol are given over 20 minutes and may be repeated every 8 hours.
- **Before Mannitol is adminstered , it is essential to :**

1. Exclude an intracranial hematoma.
2. Check the renal function.

(b) Frusemide : 40-80 mg IM is an alternative to mannitol.

(c) Corticosteroids : given empirically.

6. Physiotherapy : to joints and massage to muscles.
7. Frequent change of posture : to avoid bed sores.

Causes of deterioration of the patient

1. Brain edema : leading to increased ICT.
2. Airway obstruction &/OR Hypoventilation : leading to brain swelling and increased ICT.
3. Intracranial hematoma. **4. Fever.** **5. Overtransfusion :** by hypotonic fluids.
6. Epilepsy : if not accompanied by convulsions, it is difficult to differentiate epilepsy from an intracranial hematoma.

[5] Surgery to evacuate an intracranial hematoma : Operation :

1. Anaesthesia : The operation is done under **General Anaesthesia.**
2. Position : supine with head raised slightly to reduce venous bleeding.
3. A formal craniotomy is done. 4 or 5 burr holes are done & connected by a saw.
4. Skin & temporalis are not dissected from bone.
The whole osteoplastic flap is turned down, thus exposing the dura.
5. If an extradural hematoma is present, it can be removed by :

- Suction and the middle meningeal artery is coagulated.
- Underrun by a stitch.
- Packing the middle meningeal artery at foramen spinosum with bone wax.

6. If a subdural hematoma is found, the dura is opened in a cruciate fashion to allow rapid decompression of the brain.
7. A drain is placed & the flap is returned to its place and fixed by suturing temporalis fascia and skin.
- **Prophylactic anticonvulsant drugs are prescribed for 6 weeks to guard against epileptic fits.**

Late Complications Of Head Injury

1. Chronic subdural hematoma.
2. Post-traumatic headache : Patients with serious head injury usually have some residual symptoms as headache , giddiness , impaired sleep & defective concentration.
3. Post-traumatic epilepsy.
4. Post-traumatic hydrocephalus : This may need shunt operation.

Chronic subdural hematoma

Incidence & Etiology :

- This complication is especially liable to occur in elderly persons and alcoholics secondary to a slight blow to the head which may pass unnoticed.The sudden displacement of the brain causes rupture usually of the superior cerebral veins as they pass to the venous sinuses.
- The cerebral atrophy in elderly persons & alcoholics makes the displacement of the brain easiest.
- The result will be a collection of blood in the subdural space.
- **This hematoma is bilateral in 50% of cases.**

Clinical Picture :

1. The interval between the trauma and the symptoms : varies between weeks to months.
2. The symptoms are vague and consist of : Chronic headache and Mental apathy.
3. Physical signs : may be absent. Pupillary changes are late and denote impending conization.
4. The condition may be diagnosed as psychosis or cerebrovascular accident.

Investigations :

1. Fundus examination. **2. CT scan.**
3. Lumbar Puncture : This will reveal fluid at a low pressure with increased ptn content , often stained yellow but there are no cells.

Treatment :

- Is by evacuation through burr holes.
- If the blood is thick, bi-frontal burr holes are done and the subdural space is washed with saline.

Intracranial Abscess

Intracranial infection can be divided into :

1. Brain abscess.
2. Subdural abscess.
3. Extradural abscess.
4. Septic meningitis.

Brain abscess

Routes of Infection :

1. Local extension from adjacent infection e.g. otitis media and sinusitis.
2. Direct inoculation e.g. trauma.
3. Hematogenous.

Pathology : The abscess passes in to 3 stages :

1. Acute stage : Acute encephalitis with swelling and edema of the affected part. No pus is formed at this stage.
2. Subacute stage : Pus is formed and is surrounded by a thin wall. It gets localized.
3. Chronic stage : Thick wall with pus inside.

Clinical Picture :

[A] Acute abscess will present with :	[B] Chronic abscess will present with :
1. Picture of infection and toxaemia 2. Picture of increased ICT. 3. The commonest symptom is progressively severe headache resistant to analgesics.	1. Picture of increased ICT. 2. Focal signs of brain lesions according to the lobe affected.

Investigations : Are those of an intracranial space occupying lesion.

Treatment :

[A] Medical treatment :

1. Antibiotics.
2. Corticosteroids : reduce cerebral edema.

[B] Surgical treatment :

Factors that favour aspiration	Factors that favour excision
1. Multiple abscesses. 2. Deeply seated abscess. 3. A critical location (e.g. motor or speech area). 4. Poor general condition of the patient.	1. Multilocular abscess. 2. A superficial abscess. 3. The presence of a foreign body.

Extradural Abscess

Pathology : An abscess collected between the dura and skull secondary to osteomyelitis. It is always due to local spread of infection.

Clinical Picture :

Localized pain and tenderness over the part of the skull affected (like osteomyelitis else where).

Treatment :

1. Antibiotics. 2. Drainage by multiple burr holes over the region of the abscess.

Subdural Abscess

Pathology : It is usually due to septic thrombophlebitis of dural sinuses from local spread of infection.

Clinical Picture :

1. Signs of toxemia.
2. Marked signs of increase ICT due to blockage of the brain sinuses.

Treatment :

1. Antibiotics. 2. The abscess is drained through a burr hole.

Hydrocephalus in children

Pathophysiology :

Hydrocephalus usually results from an obstruction in CSF pathway , and only rarely , from overproduction of CSF.

Clinical Picture :

[A] If hydrocephalus occurs before closure of the skull sutures , the infant presents with macrocephaly , tense non-pulsatile anterior fontanel & separate cranial sutures. The eyes show the sunset appearance.
[B] If the sutures have already closed , the child usually presents with intracranial pressure.

Investigations :

1. X-Ray of the skull : demonstrates separation of sutures in infants , and the beaten silver appearance in older children.
2. Cranial ultrasonography.
3. CT and MRI.

Treatment :

- **Ventriculo-peritoneal shunt** OR **Ventriculo-atrial shunt.**

Spina Bifida

Definition :

A deficiency of the vertebral neural arches through which a tumor formed by maldeveloped cord or membranes protrudes.

Varieties :

1. Myelocele : The central canal of spinal cord opens upon the skin surface in the sacral region (incompatible with life).
2. Meningomyelocele : Dilatation of the membranes forms a cyst , the cord is attached to its dorsal wall and the nerves pass through the cyst.
3. Meningocele : A cyst formed of spinal membranes posterior to the cord which is not affected.
4. Spina bifida occulta : No tumor occurs , except perhaps a lipoma or a dermoid with long hair.
5. Syringomyelocele: a rare form where the central canal is dilated.

Clinical Picture :

1. Swelling in the median line of the spine usually over the lumbo-sacral region. The swelling is translucent and fluctuant (it is opaque in meningomyelocele).
2. Pressure on the swelling leads to bulging of the anterior fontanel.
3. The edges of the gap in the vertebral laminae can be felt.
4. Meningomyelocele may be accompanied by paralysis of leg muscles , talipes pes cavus and incontinence.

Treatment :

Operative treatment with an attempt to replace the cord and nerves and suture the membranes and skin over them.

Chest Injuries

Chest Injuries

I. Incidence and importance :

- In civil practice, chest injuries are mainly due to car accidents and stab wounds.
- In war, they are very common, accounting for 10% of all casualties and 25% of all fatalities
- Fatal internal lesion may occur as ruptured heart, aorta or trachea with minimal external signs.
- Saving such cases will depend mainly on proper first aid to save life by non-specialist and then rapid transfer to specialist in reasonable condition.

II. Etiology and Mechanism of injury :

1. **Blunt injuries.** e.g. fall from a height, or motor car accidents.
2. **Penetrating injuries.** Caused by stabs, or gunshots.
3. **Blast injuries.** To the chest.

III. Types :

- **Closed :** from crushes. **OR** **Open :** from stabs and bullets.
- Any of these is either pure Thoracic or Throraco-abdominal injury.

N.B. A stab wound or a bullet injury to the lower chest region may cause an associated abdominal injury.

IV. The following injuries may occur :

1. Fractured ribs : simple fractures and flail chest.
2. Hemothorax.
3. Pneumothorax.
4. Contusion or laceration of the lung.
5. Diaphragmatic injuries.
6. Oesophageal injuries.
7. Cardiac injuries.
8. Injuries to the major blood vessels.

V. Dangers that may cause death :

(A) Respiratory Insufficiency : From :

- **Upper airway obstruction.**
- **Effect on gaseous exchange due to :**

1. A flail chest due to multiple rib fractures (Stove-in-chest) causes **paradoxical movement** during respiration. The flail area is sucked during inspiration and bulges out during expiration. This will result in mediastinal flutter & pendular respiration.
2. Loss of negative intra-pleural pressure due to accumulation of air or blood. In tension pneumothorax the mediastinum is shifted to the other side. In open pneumothorax, the collapsed lung exchanges its air with that of the opposite lung during respiration **(pendulum movement).** Even the whole **mediastinum flutter** from side to side.
3. Pain reduces range of respiratory movement.
4. Blood and retained secretion in air passages.

(B) Circulatory insufficiency : From :

1. Blood loss.
2. Heart failure from cardiac contusion or rupture.
3. Effects on venous return due to :

- Mediastinal flutter diminishes venous return and cardiac output.
- Loss of the negative intra-thoracic pressure.
- Displacement of the mediastinum.

VI. Clinical Picture :

- **Life saving first-aid treatment is started as early as possible. Only when patient is stabilized, detailed examination and X-ray are done.**

1. History of trauma.
2. Local Chest Lesions, specific clinical picture of each injury.
3. General examination may reveal associated injuries e.g. abdominal injuries.

VII. Investigations : Are only done after resuscitation :

1. Urgent blood urea, urine and blood examination for diabetes, Hb%, hematocrete and blood gases.
2. Chest X-ray may show : foreign body, rib fracture, open pneumothorax, tension pneumothorax, hemothorax, hemopneumothorax, or wide mediastinum in aortic rupture.
3. Diagnostic needle aspiration.
4. Specific investigations may be needed : ECG, bronchoscopy, aortography, cardiac catheterization.
5. Frequent monitoring of : BP, CVP, Pulse, respiration, urine output, blood loss from any source.

VIII. Treatment :

[1] First Aid :

- **Life saving first-aid treatment is started as early as possible. Only when patient is stabilized, detailed examination and X-ray are done.**
- **Taking care of : A, B, C, D, and E.**

(A) Airway :

1. Airway is cleared by suction or by rolling patient on the side and cleaning his mouth by swabs. Patient airway is maintained by extending neck and pushing mandible forward.
2. Emergency tracheostomy is done if endotracheal tube is unsafe as in severe facial, oral or cervical injury. (Tracheostomy is also done to replace endotracheal tube if assisted ventilation is still needed after 3 days).

(B) Correcting respiratory insufficiency :

1. Flail chest is immobilised by cotton pad and adhesive plaster from sternum to spine to stop paradoxical movements.
2. Hemothorax or hemopneumothorax with severe dyspnea necessitates underwater seal drainage in 5th space in midaxillary line.
3. Sucking wound with open pneumothorax is sealed immediately by vaseline gauze and adhesive plaster and then underwater-seal drainage is done.
4. Tension pneumothorax is relieved by putting needle in pleural cavity until underwater-seal drainage is done in the second space anteriorly.
5. Endotracheal tube and assisted ventilation is needed in severe respiratory insufficiency not relieved by previous measures.

(C) Correcting circulatory insufficiency :

1. Restoring blood volume by blood transfusion & fluids.
2. Pericardiocentesis is life saving in cardiac tamponade.
3. Heart failure from cardiac contusion or rupture or from overtransfusion is managed as usual with digoxin, diuretics and fluid restriction.

Sever cases are better transferred to ICU.

[2] Definitive Treatment :

- Conservative treatment is continued.
- Each condition is treated accordingly.

[3] Urgent thoracotomy :

- **Indications :**

1. Massive bleeding more than 200 ml/hour.
2. Clotted hemothorax. This is diagnosed by failure of the tube to drain the blood. These patients need thoracotomy for evacuation of the clot and decortication of both the lung and chest wall.
3. Loculated hemothorax. The tube partially evacuates the blood.
4. If the hemothorax is associated with extensive wounds, severe lung trauma or foreign bodies.

Underwater seal

Definition :

Special method to drain pleural cavity to intercostal (chest) tube which ends under surface of water in special bottle.

Advantage :

It allows drainage of pleural contents (blood, air or pus). As its end is under surface of water, air can't enter pleural space. Thus gradual lung expansion occurs as pleural contents are evacuated.

Indications :

Pneumothorax, Hemothorax, empyema, after thoracotomy.

Technique :

Sterile intercostal (chest) tube is used. Its end which will be passed to pleural cavity is bevelled doubly to make it pointed, and then side hole is made just near tip.

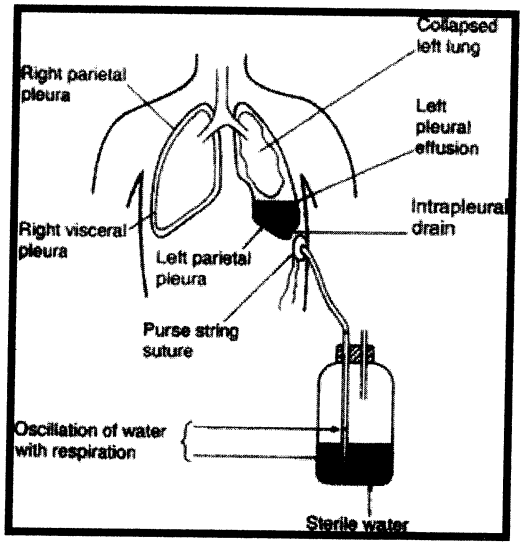
Drainage Site is infiltrated with local anesthetic :

- In hemothorax, hemopneumothorax, and empyema or after thoracotomy, drainage is done through 5th space in midaxillary line (dependent).
- Small stab is done, and the tube is pushed up in pleural cavity.

- Other end of tube is passed under surface of water in special bottle. Bottle should be always **50cm** (at least) below patient's chest so that intrathoracic negativity cannot suck its contents.
- Short column of water corresponding to intrathoracic negativity rises in the tube and oscillates with respiratory movements.

- Drainage must be helped by chest exercises.
- As lung expands, pleural contents are expelled into chest tube and nothing returns, so that complete expansion is rapidly achieved.

- Chest X-ray is done daily to follow lung expansion.
- During changing bottle or moving patient to x-ray department, chest tube must be clamped until its end is again under surface of water and the bottle 50 cm below patient's chest, to avoid sucking air or water into pleural cavity. For this reason, 2 clamps should be always besides patients.



Removing chest tube :

- **Is done when :**

1. All pleural contents are expelled
2. Lung is fully expanded proved by X-ray.

- **Chest tube is pulled out & chest wound is sealed immediately by stitching & putting adhesive plaster.**

Rib fractures

Incidence :

They are common in open or closed chest injuries.

Etiology :

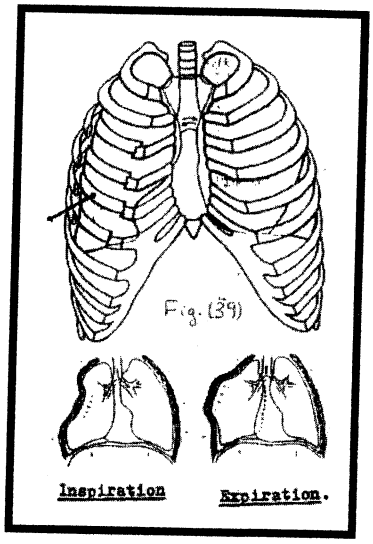
1. Direct trauma e.g. a blow or crush, may produce a fracture at any site. Visceral injury is common as the broken ends are driven inwards.
2. Indirect trauma by compression of the chest (burst fractures). The rib usually breaks at the angle, and since the broken ends are driven outwards visceral injury is uncommon.
3. Muscular violence, e.g. during violent sneezing, coughing or lifting a heavy weight, may cause fracture of the anterior segment of a rib in old people (senile osteoporosis).

Types :

- Fractured ribs may be simple or flail segment fractures.
- It may be open or closed.

Other injuries may be associated :

- Chest injuries : see before.
- Abdominal injuries e.g. spleen, liver or kidney.



Clinical picture :

[A] Simple fracture of ribs :

- Pain, tenderness and may be crepitus at the site of the fracture.
- The severe pain associated with multiple fractures may limit the respiratory expansion especially in elderly persons.
- C/P of underlying hemothorax or pneumothorax.

[B] Other cases may be complicated with :

1. Flail chest : Bad respiratory effects of flail chest are due to :
(a) Insufficient respiratory movements of related lung.
(b) Pendulum respiration & Mediastinal flutter.
2. Open pneumothorax.
3. Tension pneumothorax
4. Hemothorax or hemopneumothorax.
5. Associated abdominal injury of spleen, liver, kidney.

Treatment :

[A] Simple fracture ribs : Relief of pain to allow the patient to expand the lungs properly :
1. Strong analgesics as pethidine or non-steroidal anti-inflammatory drugs (NSAIDs) are used.
2. Intercostal nerve block may be required for persistent pain.
3. In the elderly, an elastic corset may be useful.

[B] Flail Chest :
1. If the flail segment is small : External immobilization of this segment by a cotton pad and adhesive plaster is enough.
2. In patients with sever paradoxical respiration : Introduce an endotracheal tube and start intermittent positive pressure breathing (IPPB) which will lead to internal stabilization of the flail segment.
3. If there is an indication for thoracotomy : The fractured ribs are secured by stainless steel wires or by special nails.

[C] Treatment of Associated chest or abdominal injuries.

Hemothorax

Definition : Blood collection in pleural cavity. Air may coexist, i.e. hemopneumothorax

Causes :

[A] Traumatic	[B] Spontaneous
1. Chest injuries open or closed, with injured intercostal vessels (systemic) or lung (Pulmonary). 2. Postoperative.	1. Tumors of lung, pleura, mediastinum. 2. Ruptured aortic aneurysm. 3. Blood disease.

Types : Open or closed.

Source of Bleeding :

1. Usually the intercostal or internal mammary vessels.
2. Pulmonary vessels bleeding from a lung laceration is not profuse because the pressure in the pulmonary circulation is low, and when the lung is compressed by the hemothorax, the bleeding vessels are compressed.

Pathological sequelae :

1. The respiratory and cardiac movements defibrinate the blood, so that the collection remains fluid in the majority of cases.
2. Blood in the plural sac is an irritant. It excite the formation of an effusion rich in proteins.
3. In neglected cases a fibrin layer is deposited on both layers of the pleura, later, this deposit is transformed to a fibrous layer. The end result will be a collapsed lung and deformity of the chest wall with crowding of the ribs.
4. Blood is an excellent culture medium and infection is common leading to empyema.
5. In traumatic cases there is commonly an associated pneumothorax i.e. hemopneumothorax.

Clinical picture :

- The patient complains of chest pain and dyspnoea.
- Clinical picture of hypovlemia. (Discuss).
- **Chest examination :** Reveals evidence of fluid in the pleural space; diminished lung expansion, dullness and there may be shift of the mediastinum to the opposite side in case of massive hemothorax.

Investigation :

Plain chest X-ray :

- If the hemothorax is less than 500 ml, it will lead only to obliteration of the costophrenic angle.
- Larger amounts will lead to an opacity rising to the axilla.
- Hemopneumothorax (a combination of blood and air accumulation in the pleura) shows as a transverse air–fluid level and lung collapse.

Treatment :

[A] Early cases :

- If the hemothorax is not massive it can be aspirated.
- If it is massive, insert an intercostal tube. It should be kept until no more drainage occurs and the lung is fully expanded.

[B] Late management of complications :

- Clotted hemothorax = Decortication of lung and chest wall.
- Empyema : See later.

[C] Indications for thoracotomy :

1. Massive bleeding more than 200 ml/hour.
2. Clotted hemothorax. This is diagnosed by failure of the tube to drain the blood. These patients need thoracotomy for evacuation of the clot and decortication of both the lung and chest wall.
3. Loculated hemothorax. The tube partially evacuates the blood.
4. If the hemothorax is associated with extensive wounds, severe lung trauma or foreign bodies.

Pneumothorax

Etiology :

1. Traumatic pneumothorax : by either blunt or penetrating injuries.

2. Iatrogenic pneumothorax is due to :

(a) Positive pressure ventilation that is complicated by rupture of alveoli (barotraumas).

(b) During insertion of a central venous line in neck.

3. Spontaneous pneumothorax is due to : Rupture of an emphysematous bulla, cystic lung or small subpleural tuberculous cavity.

Types :

- **There are three types of pneumothorax :**

1. Simple pneumothorax : due to entrance of limited amount of air in pleural cavity with an intact thoracic wall with no general effects.

2. Open pneumothorax : Sucking chest wound. Occurs when pleural space communicates freely with exterior, in either open chest injury with sucking wound or closed injury with tracheobronchial or lung rupture.

3. Tension pneumothorax : Occurs when pleural space communicates with exterior by valvular wound which allows air in, and not out of pleural space. It causes severe dyspnea and cyanosis.

It produces the flowing bad effects :

[A] Open pneumothorax :

1. Collapse of related lung with impaired gas exchanges.
2. Pendulum respiration i.e., oscillation of air between the two lungs. During inspiration, normal lung expands while on the other side air is sucked in pleural cavity and related lung collapse allowing normal lung to suck air from it. During expiration, normal lung collapses and expels some air in affected lung which expands slightly on expense of displacing some air out from pleural space.
3. Mediastinal flutter = Heart and mediastinum move from side to side with respiratory movement of normal lung, disturbing heart action.
4. Intrathoracic negativity is lost on affected side and reduced on normal side, interfering with venous return and cardiac filling.

[B] Tension pneumothorax :

1. Collapse of related lung with impaired gas exchange.
2. Collapse of other lung from pushed mediastinum, impairing gas exchange in other lung.
3. Mediastinal shift disturbs heart action.
4. Intrathoracic negativity is lost, interfering with venous return and cardiac filling.

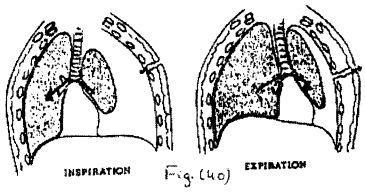


Fig. (40)

Clinical picture :

[A] Simple pneumothorax :

- Chest pain and slight dyspnea.
- There is diminished air entry, hyperresonance but there is no mediastinal shift.

[B] Open pneumothorax :

- Chest pain , dyspnea & cyanosis.
- There is absent breath sounds with hyperresonance on affected side and trachea is central.
- Sucking wound produces characteristic noise of in-out air movement.

[C] Tension pneumothorax :

- Severe dyspnea & cyanosis .
- There is absent breath sounds with hyperresonance on affected side, and trachea and apex-beat (mediastinum) are pushed to other side.
- Respiratory arrest may occur.

Treatment :

[A] Simple pneumothorax :

- If the amount of air is small and the patient is not dyspneic, the patient is treated conservatively until spontaneous absorption of the pneumothorax takes place.
- If the patient is dyspneic, an intercostal tube is inserted until expansion of the lung becomes complete.

[B] Open pneumothorax : (Emergency)

- Immediate application of an adhesive external dressing to stop movement of air through the defect.
- The wound is then repaired in theatre, and an intercostal tube is inserted to drain the air in the plural cavity.

[C] Tension pneumothorax : (Emergency)

- A needle should be immediately inserted in the second intercostal space to allow decompression of the pneumothorax. Later, an intercostal tube connected to an underwater seal is inserted.
- **Continuous bubbling of air through the intercostal tube denotes the possibility of a bronchopleural fistula.**

Empyema

Definition :

An empyema is a collection of pus in the pleural cavity.

I- Acute Empyema

Suppurative pleurisy in which there is expansion of the lung after evacuation of pus.

Routes of infection :

1. Direct : open wound as in post-traumatic or postoperative empyema.
2. Local spread : form rib, lung (pneumonia), oesophagus or diaphragm.
3. Blood spread : rare e.g. pyemia.

Pathology : Four types :

1. Pneumococcal empyema : follows lobar pneumonia (metapneumonic). Pus is thick, greenish with heavy fibrin deposits.
2. Streptococcal empyema : occurs during bronchopneumonia (synpneumonic empyema). Empyema is generalised, pus is thin, and localisation is late.
3. Staphylococcal empyema : Spread from Staph. Pneumonia in children.
4. Putrid empyema : due to rupture of lung abscess.

Clinical Picture :

- Chest pain, dyspnea and cyanosis and picture of toxemia.
- **On examination :** Signs of pleural effusion, with stony dullness, absent breath sounds, diminished chest movements and displacement of mediastinum.

Investigations :

1. X-ray.
2. Aspiration.

Treatment :

[A] General : Antibiotics, tonics and oxygen.
[B] Local evacuation of the pus by :
1. Repeated aspiration : Used in early stages with thin pus and in diffuse empyema.
2. Closed drainage : by an intercostal tube (self retaining tube) connected to an underwater seal. This is used when aspiration fails due to thick pus blocking the aspiration needle.
3. Open drainage : by rib resection : It is indicated when full localization has occurred.

II- Chronic Empyema

May be non specific or specific e.g. T.B.

Tuberculous empyema

Tuberculous infection of the pleura may occur as a complication of :

1. Rupture of a tuberculous cavity.
2. Pleural effusion.
3. Operations on tuberculous lungs.

Treatment :

1. In the absence of secondary infection :

- Repeated aspiration.
- Anti-tuberculous treatment.

2. Cases with secondary infection :

- Prolonged drainage until the secondary infection is overcomed followed by decortication.

Chronic non-specific empyema

Types :

[A] Open chronic Empyema :

- **(Chronic empyema sinus) due to improper treatment of acute empyema :**

1. Faulty drainage : Too early drainage (collapse of lung), Too late drainage (rigid pleura), Too high (independent drainage) or Too low (opening blocked by diaphragm).
2. Inadequate post-operative care e.g. early removal of the tube.
3. Presence of foreign body.
4. Underlying disease, e.g. osteomyelitis of rib, T.B. or actinomycosis of pleura, lung abscess, bronchiectasis, bronchopleural fistula.

[B] Closed chronic empyema : As encysted empyema and recurrent empyema.

[C] Bronchopleural fistula.

Clinical Picture :

1. General ill-health with anaemia and clubbing of the fingers.
2. Chronic sinus in the chest wall discharging pus.
3. Rarely, the empyema remains latent until discovered by X-ray examination, or until it perforates an intercostal space to form a subcutaneous abscess **(empyema necessitanes).**
4. In long standing cases, Progressive fibrosis causes contraction and rigidity of the chest wall with crowding of the ribs, elevation of the diaphragm, displacement of the mediastinum to the affected side and scoliotic deformity of the spine.

Treatment :

- **An attempt is made to obliterate the cavity by :**

1. Adequate drainage established at a suitable level and followed by proper lavage and physiotherapy cures 70% of cases within 3 months.
2. Decortication Which is complete excision of the thickened visceral pleura which forms a tight **"peel"** around the lung so that the freed lung can expand and obliterate the cavity.

Cardiac arrest

Causes :

1. During anaethesia and surgical operations (a potential danger).
2. Myocardial infarction.
3. Electrocution.
4. Near-drowning.
5. Asphyxia.
6. Gas poisoning.
7. Severe trauma.

Pathogenesis :

Cardiac arrest may be due to asystole or to ventricular fibrillation, but the distinction is impossible until an ECG is obtained or the heart is exposed.

Two chief factors predispose to cardiac arrest :

I- Inadequate venous return caused by :

1. **Massive haemorrhage.**
2. Vasomotor paralysis e.g. general Anaesthetic agent or spinal anaesthesia.
3. Vascular obstruction by massive pulmonary embolism and air embolism.

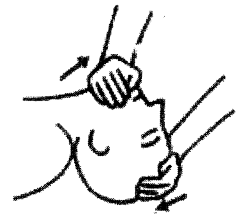
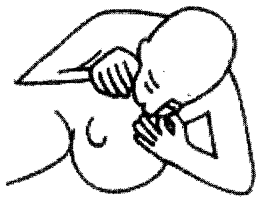


Fig (41)



II- Myocardial depression by :

1. Anoxia due to respiratory obstruction, hypotension or **coronary occlusion.**
2. Co_2 retention, particularly when suddenly relieved.
3. Irritating drugs e.g. chloroform & adrenaline.
4. Vagal inhibition e.g. by carotid sinus syndrome.
5. Electrolyte imbalance e.g. acidosis and excessively high or low potassium.
6. Trauma, such as coarse manipulation during surgical operations.
7. Hypothermia, Myocardial disease.

Clinical picture :

- **The cardinal signs of cardiac arrest are :**

1. Absent carotid pulse.
2. Absent or gasping respiration.
3. Dilated pupils.

- **The stethoscope has no place in the diagnosis of cardiac arrest.**

Treatment :

The brain can only tolerate 3 minutes of complete circulatory arrest and unless the circulation is restored within this period, death or permanent neurological damage will result.

- **The proper management consists of three phases :**

I- External cardiopulmonary resuscitation :

- By direct mouth-to-mouth breathing and closed cardiac massage.

The aim : Is not to restart the heart but to provide an artificial circulation and ventilation until appropriate facilities are available.

Technique :

1. The patient is laid flat on a firm surface and the legs are raised to increase the venous return to heart.
2. The head is tilted back with one hand to ensure a clear airway.
3. The nostrils are closed with the other hand while the mouth is applied to the patient mouth to fill his lungs with expired air at a rate of 10-15 times /minute.
4. At the same time, a second person administers external cardiac massage by compressing the lower third of the sternum against the spine at a rate of about 60 times/minute.
5. The efficiency of ventilation and massage must be monitored by observing chest expansion and palpating the femoral pulse.
6. The patient should then be transferred to hospital as soon as possible with direct mouth-to-mouth breathing and cardiac massage continued.

II- Restoration of normal heart action (Once in hospital) :

[1] An endotracheal tube is passed and the lungs are ventilated with oxygen.

[2] A venous cannula is inserted and suitable infusions are given.

[3] Electrocardiographic monitoring is applied to :

i. Differentiate cardiac asystole from ventricular fibrillation. ii. Watch for signs of recovery.

(A) If cardiac asystole is present :

1. Massage is continued.
2. Intracardiac injection of adrenaline or IV calcium chloride.
3. Either sinus rhythm or fibrillation may occur.
4. If there is no response open cardiac massage should be done.

(B) If ventricular fibrillation is present or develops after a systole :

1. External defibrillator is applied, and a 0.2 second of 800 volt shock is given.
2. If unsuccessful the procedure is repeated.
3. Defibrillation either restores sinus rhythm or induces cardiac asystole.
4. If external Defibrillation is not available or unsuccessful open cardiac massage and internal defibrillation should be carried out.

[4] Open cardiac massage :

Indication : 1. The chest is already opened. 2. If closed massage has been unsuccessful.

Method :

1. The chest is entered through the left fifth intercostal space.
2. The pericardium is widely opened to permit bimanual massage with one hand above and the other below the ventricular mass.
3. The massage is continued at a rate of 60-80 / minute until the cardiac tone returns and the heart becomes smaller, firmer and pinker.
4. If the tone does not return with massage, calcium chloride is injected IV.
5. In fibrillation cases, procaineamide is injected IV to reduce cardiac irritability and internal defibrillation is performed.
6. If cardiac asystole persists in spite of adequate myocardial tone an injection of adrenaline or isoprenaline into the right ventricle often restores the normal rhythm.

III- Subsequent treatment :

Following restoration of normal rhythm, the patient is kept under close observation in an ICU the following measures are instituted :

1. The BP is kept over 90 mmhg by vasopressors & transfusion.
2. If the chest has been opened it is left opened for 1/2 hour to watch for re-failure of the heart.
3. Cardiac stimulants, e.g. noradrenaline, Calcium chloride and digitalis may be administered as needed.
4. Hypothermia and dehydration therapy with mannitol or concentrated glucose are induced if return of consciousness is delayed.
5. Infusions of sodium bicarbonate solution are given to correct the metabolic acidosis produced by anoxia and blood transfusion.
6. Hyperkalemia is often present & should be treated with calcium chloride or glucose & insulin.
7. Anuria and renal damage should be anticipated and prevented by adequate measures.
8. The cause of cardiac arrest must be investigated and treated.

Termination of CPR :

This is a grave medical decision. A chance of 1 hour at least should be given to all patients regardless the condition. **Continuation for more than hour depends on certain criteria :**

1. Reaction of the pupils : A fixed dilated pupils for 15-30 minutes indicates cerebral death.

2. The level of consciousness : muscular movements and spontaneous breathing.

3. A flat EEG : also indicates cerebral death unless the patient had received large doses of cerebral depressants as barbiturates.

Post-operative pulmonary complications

Predisposing factors (PF) :

Preoperative PF	Operative & post operative PF
1. Age : extremes of age are more liable to complications. **2. Sex :** males are more predisposed to respiratory problems. **3. Smoking.** **4. Chronic bronchitis.** **5. Dehydration.**	**[A] Anaethesia :** 1. Prolonged unconsciousness. 2. Trauma to tracheobronchial tree. 3. Premedication with atropine. 4. Inhalation of blood or vomitus. **[B] Nature of operation :** 1. Thoracic and upper abdominal operations. 2. Malignant disease. **[C] Abdominal distension :** e.g due to paralytic ileus. **[D] Inhibition of cough reflex by :** 1. Pain. 2. Heavy narcotics.

Types :

1. Respiratory obstruction. 2. Bronchitis e.g. exacerbation of preexisting one.
3. Atelectasis : commonest complication due to retention of secretion.
4. Lung abscess complicating :
   (a) Inhalation. (b) Bronchopneumonia. (c) Atelectasis.
5. Bronchopneumonia : complicating retained secretion in chest.
6. Bronchiectasis : complicating bronchopneumonia & atclectasis.
7. Empyema. 8. Pulmonary embolism (discuss in short).
9. Mendelson's syndrome. 10. Adult respiratory distress syndrome.

Pathophysiology of postoperative pulmonary complications :

- After upper abdominal surgery, the vital capacity of the patient's lungs often drops to as low as 25% of its preoperative value. If the ratio drops more, respiratory failure may occur.
- **The situation will be exacerbated by :**

1. Postoperative distension and ileus.
2. The lungs of a smoker will be less able to cope with the reduction in vital capacity.

Prevention:

Preoperative	Operative		Post-operative
	Anesthesist avoids	**Surgeon avoids**	
1. Prohibit smoking. 2. Correct dehydration. 3. Breathing exercises.	1. Cyanosis. 2. Excessive atropinization. 3. Deep anesthesia. 4. Excessive narcotics. 5. Vomiting.	1. Rough manipulation. 2. Undue prolongation. 3. Tight bandage. 4. Tight strapping of chest & abdomen.	1. Avoid heavy sedation. 2. Encourage deep breathing, coughing, breathing exercises & postural drainage. 3. Correct dehydration. 4. Clear secretions. 5. Early ambulation.

Post-operative atelectasis (pulmonary collapse)

Predisposing factors	
[A] Production of tenacious mucus due to :	**[B] Inhibition of the cough reflex due to :**
1. Pre-operative respiratory tract infection. 2. Premedication with atropine. 3. Prolonged ether anesthesia. 4. Inhalation of FB, vomitus or septic material. 5. Post-operative dehydration.	1. Old age and debility. 2. Excessive sedation, particularly with morphine. 3. Operations on the chest, diaphragm or upper abdomen. 4. Pain & Lack of mobility in bed.

Pathology :

- Obstruction of a bronchus by a plug of mucous is followed by absorption of air distal to the obstruction and deflation of the affected area.
- **The consequent collapse may be :**

1. Lobular : collapse of scattered areas throughout the lung.
2. Lobar : collapse of one lobe usually the lower.
3. Massive : collapse of the whole lung.

- It is precursor of most postoperative chest complications, particularly bronchopneumonia and lung abscess.

Clinical features :

Symptoms :

1. In typical cases, the onset is sudden with fever in addition to dyspnea, cyanosis and chest pain.
2. Cough is slight and the sputum is scanty and difficult to expel.
3. The condition may subside after a few days with the expectoration of a considerable amount of purulent tenacious mucous.

Signs :

1. Unexplained tachycardia.
2. A minor atelectasis produces no local signs.
3. With collapse of a segment or the whole lung the affected side of the chest is flattened and immobile with dullness on percussion, diminished breath sound and displacement of the trachea and heart towards the side of collapse. As the attack subsides tubular breathing and coarse crepitations may appear.

X-ray :

- The collapsed lobe appears as a homogeneous wedge-shaped opacity.
- Major atelectasis causes approximation of the ribs, elevation of the diaphragm and deviation of the mediastinum toward the affected side.

Prophylaxis :

[A] Preoperative :

1. Eradication of respiratory infections.
2. Prohibition of smoking.
3. Breathing exercises.
4. Avoidance of dehydration & heavy premedication, particularly with atropine and morphine.

[B] Operative : Avoidance of :

1. Trauma to the upper air passages during intubation.
2. Deep unconsciousness and vomiting under anesthesia.
3. Tight strapping and bandaging of the chest and abdomen.

[C] Post-operative :

1. Correction of dehydration.
2. Avoidance of heavy sedation.
3. Encouragement of deep breathing, and postural exercises.
4. Removal of secretions by coughing, expectoration and postural drainage.
5. Early ambulation & adequate analgesia to reduce pain with coughing and deep breathing.

Treatment : All attempts are made to expel the obstructing plug

1. Initial measures :

- Turning the patient from side to side & percussing the affected side of the chest.
- Encouraging expectoration, if necessary by nasopharyngeal catheter.
- Loosening the sputum by mucolytics, expectorants and steam inhalation.

2. Suction.
3. Antibiotics.

Pulmonary embolism (PE)

Incidence :

Between 2-3% of all hospital mortalities are due, wholly or in part, to pulmonary embolism. Pulmonary emboli originate from thrombi in the venous circulation. The less common varieties of pulmonary emboli, e.g., septic and fat emboli are less frequent and will be discussed separately.

Etiology :

All the predisposing factors which lead to DVT are important in the etiology of pulmonary embolism.

Pathophysiological sequelae :

The immediate consequences of a pulmonary embolus (PE) are related to both mechanical and humoral reflex effects.

[A] Mechanical effects : result from the passage and impaction of the clot in the pulmonary arterial tree. They are relatively insignificant until more than 25% of the pulmonary artery circulation is occluded. Cardiac output remains normal until pulmonary artery occlusion exceeds 50% (massive embolism). Underlying cardiac or respiratory insufficiency contributes significantly to premature failure of cardiac output.

The mechanical effects of a PE result in :

1. Arrhythmia as the clot traverses the right atrium and ventricle.
2. Dilatation of the bronchial arteries.
3. Decreased pulmonary blood flow which results in :
   (a) Decreased cardiac output (CO) leading to systemic hypotension and shock.
   (b) High ventilation perfusion ratio.
   (c) Myocardial hypoxia.
4. Pulmonary infarction occurs in less than 10% of PE cases. The development of an infarction depends on completeness of occlusion and effectiveness of collateral circulation.

[B] Humoral reflex effects : These are less significant than the mechanical effects and are due to the release of biologically active amines (histamine and serotonin) from the platelets.

The humoral reflex effects include :

1. Increased pulmonary artery resistance and pressure and decreased pulmonary blood flow.
2. Bronchoconstriction causing increased airway resistance and decreased compliance, hypoxemia and tachypnea.

Clinical features :

- The clinical picture of pulmonary embolism is determined by the site where the embolus is impacted.
- **Accordingly three types of embolism are recognized :**

1. Small emboli are impacted in the peripheral arterioles. They are usually silent. Recurrent small emboli will lead to pulmonary hypertension.

2. Medium sized emboli lodge in the branches of the pulmonary arteries and result in pulmonary infarction. This leads to the classical picture of severe pleuritic pain, dyspnoea and haemoptysis.

3. Large emboli are impacted either in the main pulmonary artery itself or in one of its major branches leading to massive pulmonary embolism. There is severe precordial pain, tightness in the chest, marked dyspnoea, severe hypotension and marked tachycardia. Sudden death may occur.

- It should be stressed that in many patients with pulmonary embolism, there is no clinical evidence of deep vein thrombosis in the lower limbs.
- Unexplained asthma or heart failure appearing in a hospitalized patient is very suggestive of pulmonary embolism.

Differential diagnosis :

Pneumonia, congestive heart failure and myocardial infarction.

Investigations :

1. Laboratory :

- Leucocytosis if greater than 15,000/μL is suggestive of pneumonia rather than PE.
- Enzyme pattern : Raised LDH and bilirubin, with normal SGOT are present in 30% of patients.

2. Blood gases : The most consistent finding is PaO_2 < 80 mmHg. $PaCO_2$ remains normal or hypocapnia may occur.

3. ECG : The major value is to differentiate PE from myocardial infarction. In 40% of patients with PE there are large P waves, right axis deviation and T wave inversion.

4. Chest X-ray : This is entirely normal in 50% of cases or may show the following :

- Prominent pulmonary artery (hilar shadow).
- Diminished vascular markings in the area supplied by the obstructed pulmonary artery.
- Enlarged right ventricle.
- Small pleural effusion.
- Cone-shaped density based on the pleural surface.

5. Tests to detect occult venous thrombosis. The most valuable is **duplex scan.**

6. Ventilation-perfusion pulmonary isotope scan with technetium^{99m} : Non-perfused areas show as defects.

7. Pulmonary arteriography : This is a definitive diagnostic investigation.

8. Contrast CT : This is also a diagnostic and less invasive technique.

Treatment :

[A] Minor and moderate pulmonary emboli are treated by anticoagulants.

[B] Patients with massive pulmonary embolism represent a very serious surgical emergency and should be immediately transferred to a cardiothoracic centre where urgent pulmonary arteriography is performed. If the diagnosis is confirmed, the following measures are performed :

- The cardiac catheter is left in the pulmonary artery & thrombolytic therapy should be started.
- If deterioration sets in, urgent pulmonary embolectomy is performed.

[C] Vena caval interruption to prevent recurrence of PE.

- **Indications :**

1. When anticoagulant therapy is contraindicated, or if it fails to stop recurrent emboli.
2. First PE in high risk patients.
3. History of recurrent PE.
4. Septic phlebitis.
5. Following pulmonary embolectomy.

Special types of pulmonary embolism

I- Septic pulmonary emboli

The emboli originate from septic thrombophlebitis. They tend to be small and multiple and produce multiple foci of infection in the lung.

Anticoagulants are contraindicated since they tend to promote haemorrhage in infected pulmonary foci.

Treatment involves 3 essential steps :

1. Proximal venous ligation.
2. Antibiotics in high doses.
3. Drainage of any closed infection.

II- Bone marrow and fat emboli

- This is a rare condition.
- 95% of fat emboli are associated with fractures.
- 75% of fat emboli are trapped in the pulmonary circulation.
- The fat emboli lodged in the pulmonary capillaries are acted upon by the lipase enzyme producing free fatty acids. The latter are toxic and produce disruption of the alveolar capillary membrane and destruction of surfactant leading to pulmonary oedema, pulmonary haemorrhage and alveolar collapse.

Clinical features :

- These usually begin 12-96 hours after the injury with dyspnea, tachypnea, cyanosis, crepitations and pleural friction rub.
- Cerebral symptoms as confusion, delirium or coma may occur.
- Peticheal haemorrhages on the chest wall, axilla, flanks or conjunctiva may be present.
- The temperature may rise up to 39°C.

Investigations :

1. Lipemia. Fat is also present in urine in 60% of cases.
2. Elevated serum lipase.
3. Chest X-ray reveals bilateral stippled or fluffy pulmonary infiltrates.
4. ECG reveals right sided strain.
5. Low PaO_2 with compensatory respiratory alkalosis.

Treatment :

1. Correction of the hypovolaemia.
2. Splinting and immobilization of the fracture.
3. Respiratory support with oxygen.
4. Parenteral steroids.

Mendelson's syndrome

Etiology :

Aspiration of vomitus or gastric contents during induction of anesthesia or comatosed patients.

Clinical picture :

Wheezes, cyanosis, tachycardia, tachypnea and hypotension.

Radiologically :

Widespread lung infiltration (more in the right lower lung lobes).

Blood gas analysis :

Reveals severe hypoxia

Prophylaxis :

All high risk patients should have a nasogastric tube.

Treatment :

Adequate bronchoscopic aspiration, steroids, bronchodilators and antibiotics

Adult respiratory distress syndrome (ARDS, shock lung)

Etiology :

This syndrome can occur in patients suffering from :

1. Multisystem trauma.
2. Severe abdominal or generalized sepsis.
3. Extensive burns.
4. Exposure to whole body irradiation.
5. Severe shock from any cause.
6. Lung injury.
7. Iatrogenic factors:
   (a) Non-filtered blood transfusion.
   (b) Overtransfusion of fluids.
   (c) Use of oxygen concentrations over 50%.
   (d) Massive doses of steroids
   (e) The use of a heart lung machine for open heart surgery for a long time.

Pathogenesis :

- **ARDS is supposed to be due to activation of platelets, neutrophils and macrophages leading to release of O2 free radicles which cause :**

1. Endothelial damage.
2. Edema of lung tissue.
3. Increased capillary permeability & so neutrophils & R.B.C.'s move to the inside of alveoli.
4. Impairment of O2 diffusion.

Defects occur in the three aspects of the respiratory process :

1. Defective ventilation
2. Defective perfusion
3. Defective diffusion.

Clinical Progress :

1. Initial state of shock, lactic acidosis and hyperventilation with low PaCO2, but PaO2 may be normal or slightly low. During resuscitation the pathophysiology is dependant on the basic disease.

2. Phase of hemodynamic equilibrium which may last several days. The patient may appear well and recovering with a normal chest X-ray but the PaO2 is invariably low.

3. Phase of clinical respiratory distress followed by respiratory failure with confusion and occasional petechial rash. Rising PaCO2 and falling PaO2 occur despite oxygen supplement. Chest x-ray reveals bilateral pulmonary infiltrations.

Treatment :

- The patient should be admitted to an intensive care unit (ICU) :

1. Treatment of the cause e.g. correction of shock and eradication of sepsis.

2. Respiratory support :

(a) In the stage of respiratory distress, an oxygen mask is enough to improve arterial blood oxygen.
(b) In the stage of respiratory failure (indicated by a PaO2 < 60 mmHg) endotracheal intubation and mechanical ventilation are required.